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September 2012

Advanced anatomy of lateral nasal wall For the endoscopic sinus surgeon

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For the endoscopic sinus surgeon

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Authors

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Anatomy of lateral nasal wall

Introduction:

Anatomy of the lateral nasal wall is highly complex and variable. With the popularity of endoscopic sinus surgery a through knowledge of this complex anatomy is very vital. Highly variable anatomy and paucity of standard landmark makes this region vulnerable for complications during endoscopic sinus surgery. The learning curve for endoscopic sinus surgery is made rather steep by this highly variable anatomy [1]. Study of anatomy of lateral nasal wall dates back to Galen (AD 130-201). He described the porosity of bones in the head. Davinci in his classical anatomical drawings has illustrated maxillary sinus antrum. He also described maxillary sinus as cavities in the bone that supports the cheek [2]. Highmore (1651) described maxillary sinus anatomy. Hence it is also known as antrum of Highmore [3]. It was during the 19th century that Zuckerkandl came out with the first detailed description of maxillary sinus and its surrounding anatomy. Paranasal sinuses are four air filled cavities situated at the entrance of the upper airway. Each of these sinuses are named after the skull bone in which it is located [4].

Nasal turbinates:

The turbinates are the most prominent feature of the lateral nasal wall [5]. They are usually three or sometimes four in number. These turbinates appear as scrolls of bone, delicate, covered by ciliated columnar epithelium. These turbinates sometimes may contain an air cell, in which case it is termed as a concha.

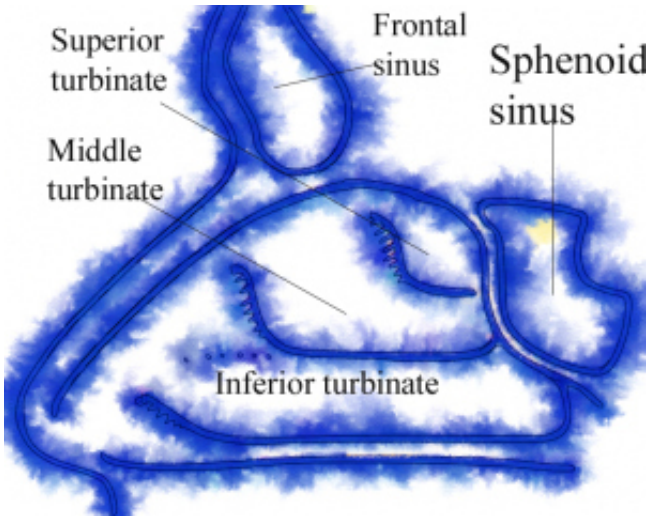
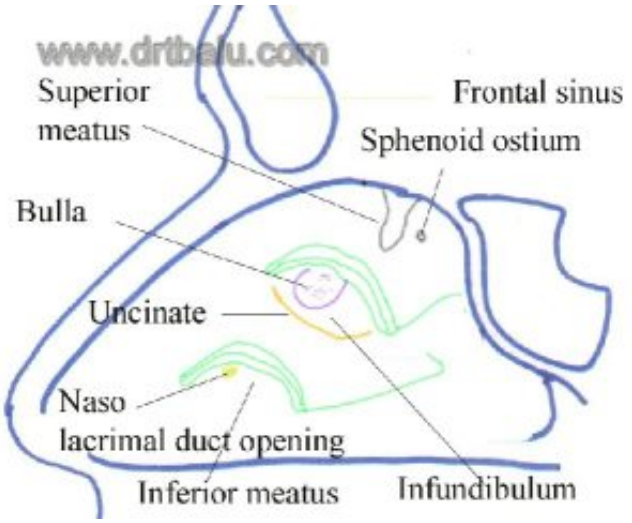


Fig. 1: Figure showing turbinates in the lateral nasal wall

These turbinates project from the lateral wall of the nose. Out of these turbinates the following are present in all individuals:

The superior, middle and inferior turbinates. A small supreme turbinate may be present in some individuals. Among these turbinates the superior and the middle turbinates are components of the

ethmodial complex where as the inferior turbinate is a separate bone. Commonly a prominence may be seen at the attachment of the middle turbinate.



lateral wall of nose after removal of turbinates

This prominence is known as the agger nasi cell. This prominence varies in size in different individuals. These agger nasi cells overlie the lacrimal sac, separated from it just by a thin layer of bone. Infact this agger nasi cell is considered to be a remnant of naso turbinal bones seen in animals. When the anterior attachment of the inferior and middle turbinates are removed, the lacrimal drainage system and sinus drainage system can be clearly seen.

The inferior turbinate is a separate bone developed embryologically from the maxilloturbinal bone.

The inferior meatus is present between the inferior turbianate and the lateral nasal wall. The nasal opening of the naso lacrimal duct opens in the anterior third of the inferior meatus. This opening is covered by a mucosal valve known as the Hassner's valve. The course of the naso lacrimal duct from the lacrimal sac lie under the agger nasi cell.

The middle meatus lie between the middle turbinate and the lateral nasal wall. The middle turbinate is part of the ethmoidal complex. The sinuses have been divided into the anterior and posterior groups. The anterior group of sinuses are frontal, maxillary and anterior ethmoidal sinuses. These sinuses drain into the middle meatus, i.e. under the middle turbinate. The middle meatus hosts from anterior to posterior the following structures:

1. Agger nasi
2. Uncinate process
3. Hiatus semilunaris
4. Ethmoidal bulla
5. Sinus lateralis
6. Posterior fontanelle

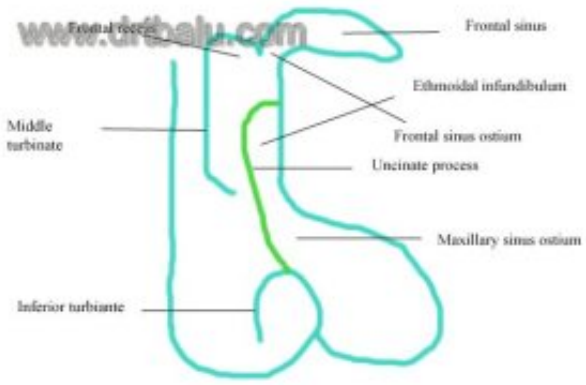
Uncinate process: actually forms the first layer or lamella of the middle meatus. This is the most stable landmark in the lateral nasal wall. It is a wing or boomerang shaped piece of bone. It attaches anteriorly to the posterior edge of the lacrimal bone, and inferiorly to the superior edge of the inferior turbinate [6]. Superior attachment of the uncinate process is highly variable, may be attached to the lamina palyracea, or the roof of the ethmoid sinus, or sometimes to the middle turbinate. The configuration of the ethmoidal infundibulum and its relationship to the frontal recess depends largely

on the behavior of the uncinate process. The Uncinate process can be classified into 3 types depending on its superior attachment.

The anterior insertion of the uncinate process cannot be identified clearly because it is covered with mucosa which is continuous with that of the lateral nasal wall. Sometimes a small groove is visible over the area where the uncinate attaches itself to the lateral nasal wall. The anterior convex part forms the anterior boundary of the ostiomeatal complex. It is here the maxillary, anterior ethmoidal and frontal sinuses drain. Uncinate process can be displaced medially by the presence of polypoidal tissue, or laterally against the orbit in

individuals with maxillary sinus hypoplasia [7]. Removing of this piece of bone is the most important step in Endoscopic sinus surgery.

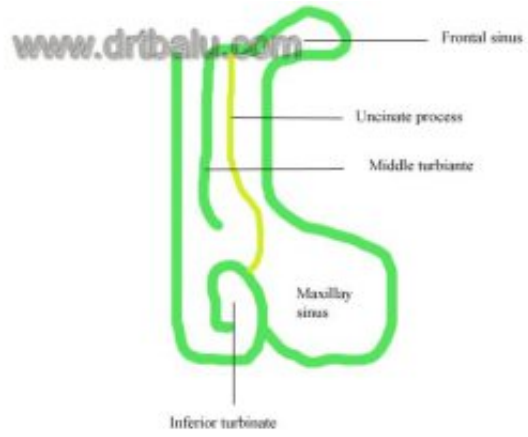
Type I uncinate: Here the uncinate process bends laterally in its upper most portion and inserts into the lamina papyracea. Here the ethmoidal infundibulum is closed superiorly by a blind pouch called the recessus terminalis (terminal recess). In this case the ethmoidal infundibulum and the frontal recess are separated from each other so that the frontal recess opens into the middle meatus medial to the ethmoidal infundibulum, between the uncinate process and the middle turbinate. The route of drainage and ventilation of the frontal sinus run medial to the ethmoidal infundibulum.



Type I uncinate insertion

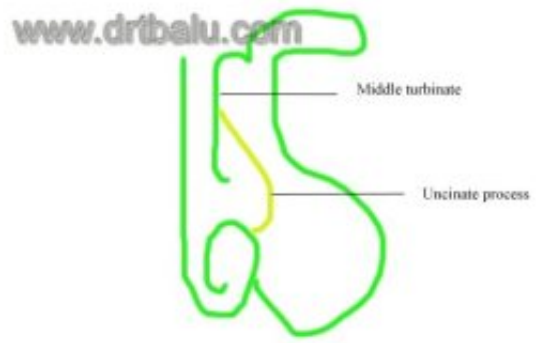
Type II uncinate: Here the uncinate process extends superiorly to the roof of the ethmoid. The frontal sinus opens directly into the ethmoidal infundibulum. In these cases a disease in the frontal recess may spread to involve the ethmoidal infundibulum and the maxillary sinus secondarily. Sometimes the superior end of the uncinate process may get divided into three branches one getting attached to the roof of the ethmoid, one getting attached to the lamina papyracea, and the last getting attached to the middle turbinate.

Type II uncinate insertion

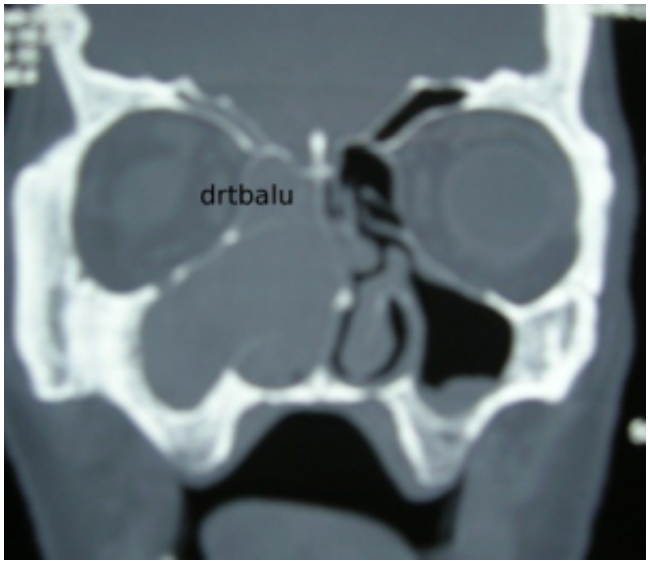


Type III uncinate process: Here the superior end of the uncinate process turns medially to get attached to the middle turbinate. Here also the frontal sinus drains directly into the ethmoidal infundibulum.

Rarely the uncinate process itself may be heavily pneumatised causing obstruction to the infundibulum.



Type III uncinate insertion



Polyp seen pushing the uncinate medially

Hypoplasia of maxillary sinus seen pushing the uncinate laterally

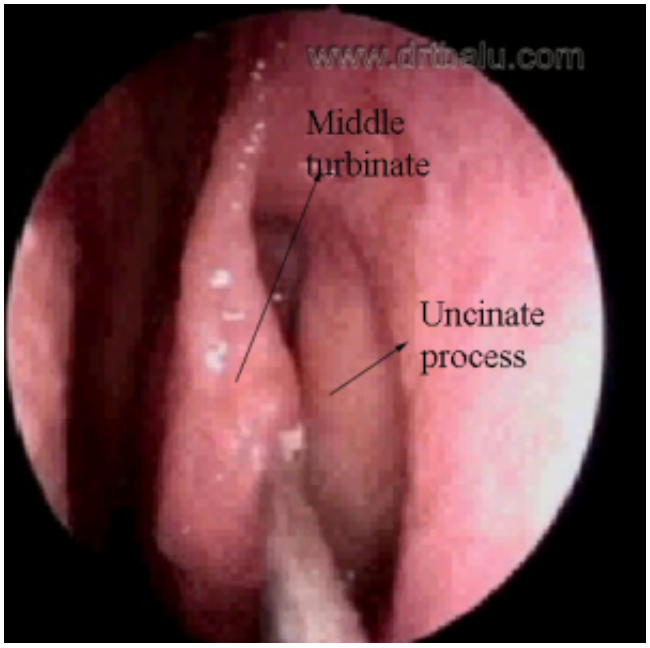


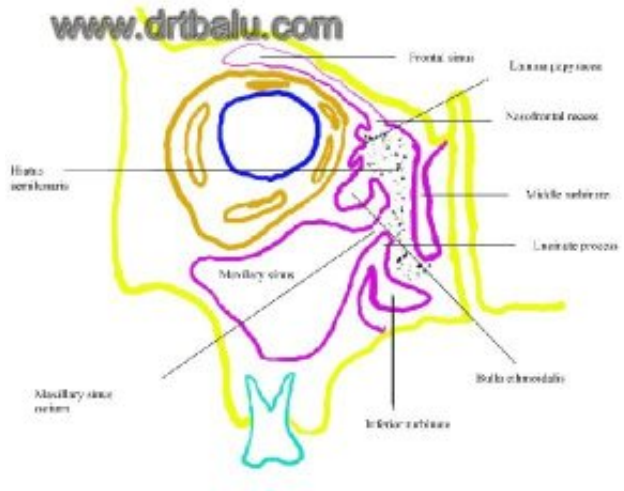
Image showing uncinate process

Removal of uncinate process reveals the natural ostium of the maxillary sinus. This is another vital landmark in the lateral nasal cavity. The superior wall of the natural ostium of the maxillary sinus is at the level of floor of the orbit.Agger nasi: This is a latin word for “Mound”. This area refers to the most superior remnant of the first ethmoturbinal which presents as a mound anterior and superior to the insertion of middle turbinate.Depending on the pneumatization of this area may reach up to the level of lacrimal fossa thereby causing narrowing of frontal sinus outflow tract. Ethmoidal infundibulum: is a cleft like space, which is three dimensional in the lateral wall of the nose. This structure belongs to the anterior ethmoid. This space is bounded medially by the uncinate process and the mucosa covering it. Major portion of its lateral wall is bounded by the lamina papyracea, and the frontal process of maxilla to a lesser extent. Defects in the medial wall of the infundibulum is covered with dense connective tissue and periosteum. These defects are known as anterior and poterior fontanelles. Anteriorly the ethmoidal infundibulum ends blindly in an acute angle.

Figure showing large agger nasi air cell

Bulla ethmoidalis: This is derived from Latin. Bulla means a hollow thin walled bony prominence. This

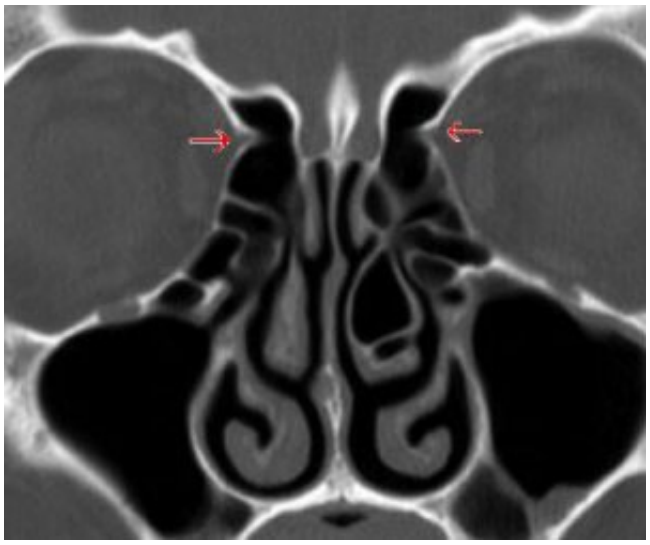
is another landmark since it is the largest and non variant of the aircells belonging to the anterior ethmoidal complex. This aircell is formed by pneumatization of bulla lamella (second ethmoid basal lamella). This air cell appears like a bleb situated in the lamina papyracea. Some authors consider this to be a middle ethmoid cell. If bulla extends up to the roof of ethmoid it can form the posterior wall of frontal recess. If it does not reach up to the level of skull base then a recess can be formed between the bulla and skull base. This recess is known as suprabullar recess. If the posterior wall of bulla is not in contact with basal lamella then a recess is formed between bulla and basal lamella. This recess is known as retrobullar recess / sinus lateralis. This retrobullar recess may communicate with the suprabullar recess. Osteomeatal complex: This term is used by the surgeon to indicate the area bounded by the middle turbinate medially, the lamina papyracea laterally, and the basal lamella superiorly and posteriorly. The inferior and anterior borders of the osteomeatal complex are open. The contents of this space are the aggernasi, nasofrontal recess (frontal recess), infundibulum, bulla ethmoidalis and the anterior group of ethmoidal air cells.This is infact a narrow anatomical region consisting of : 1. Multiple bony structures (Middle turbinate, uncinate process, Bulla ethmoidalis) 2. Air spaces (Frontal recess, ethmoidal infundibulum, middle meatus) 3. Ostia of anterior ethmoidal, maxillary and frontal sinuses. In this area, the mucosal surfaces are very close, sometimes even in contact causing secretions to accumulate. The cilia by their sweeping movements pushes the nasal secretions. If the mucosa lining this area becomes inflamed and swollen the mucociliary clearance is inhibited, eventually blocking the sinuses. Some authors divide this osteomeatal complex into anterior and posterior. The classic osteomeatal complex described already has been described as the anterior osteomeatal complex, while the space behind the basal lamella containing the posterior ethmoidal cells is referred to as the posterior ethmoidal complex, thus recognising the importance of basal lamella as an anatomical landmark to the posterior ethmoidal system. Hence the anterior and the posterior osteomeatal complex has separate drainage systems. So when the disease is limited to the anterior compartment of the osteomeatal complex, the ethmoid cells can be opened and diseased tissue removed as far as the basal lamella, leaving the basal lamella undisturbed minimising the risk during surgery.Hiatus semilunaris: Lies between the anterior wall of the Bulla and the free posterior margin of the uncinate process. This is infact a two dimensional space. Through this hiatus a cleft like space can be entered. This is known as the ehtmoidal infundibulum. This ethmoidal infundibulum is bounded medially along its entire length by the uncinate process and its lining mucosa. The lateral wall is formed by the lamina papyracea of the orbit, with participation from the frontal process of the maxilla and the lacrimal bone. The anterior group of sinuses drain into this area. Infact this area acts as a cess pool for all the secretions from the anterior group of sinuses.



Osteomeatal complex

Concha bullosa: Sometimes middle turbinate may become pneumatized. This pneumatization is known

as concha bullosa. This process of pneumatization starts either from frontal recess or agger nasi air cells. This is usually considered to be a normal variant. Sometimes this pneumatization may become so extensive that it could cause obstruction in osteomeatal complex [8].



Coronal CT showing concha bullosa

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February 2013

Anatomy of Orbit Otolaryngologist's perspective

Anatomy of Orbit

Otolaryngologist's perspective

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Author

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Abstract

A careful study of anatomy of orbit is very important to an ENT surgeon because of its proximity to the para nasal sinuses. A comprehensive knowlege of orbital and peri orbital anatomy is necessary to understand the various disorders of this region and in its surgical mangement. Current day otolaryngologists venture into other unchartered territories like orbit, lacrimal sac etc. Anatomical knowledge of this area will help otolaryngologists to avoid complications during surgical procedures involving this area. This article attempts to explore this topic from otolaryngologist's perspective.

Anatomy of orbit

Introduction:

Orbit supports the eye and ensures that this organ functions in an optimal manner. It also protects this vital structure. The shape of the orbit resembles a four sided pyramid to begin with but as one goes posterior it becomes three sided towards the apex. The volume of the orbital cavity in an adult is roughly about 30cc. The rim of orbit in an adult measures about 40mm horizontally and 35 mm vertically. The medial walls of orbit are roughly parallel and are about 25 mm apart in an adult. The lateral walls of orbit angles about 90 degrees from each other. This is actually a fixed cavity with no scope for enlargement, hence a small increase in ocular pressure can lead to disastrous consequences.

Osteology:

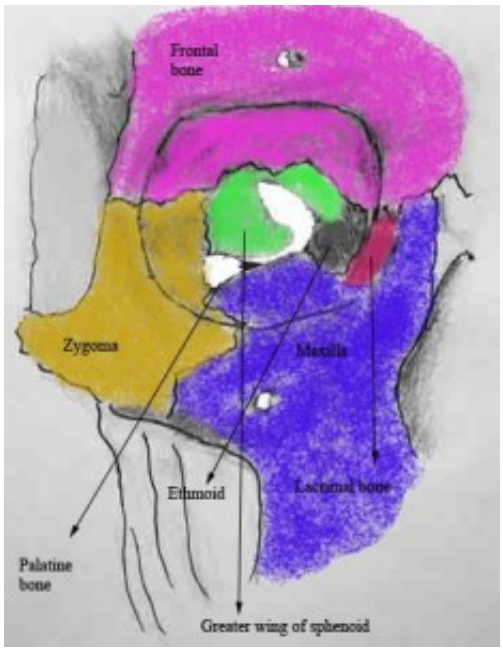
Seven bones join together to form the orbit [1] . These include:

1. Frontal bone
2. Lacrimal bone
3. Zygoma
4. Maxilla
5. Ethmoid
6. Sphenoid
7. Palate

The orbital rim is more or less spiral with its two ends overlapping medially on either side of lacrimal fossa. The inferior orbital rim is formed by the maxillary bone medially and zygomatic bone laterally.

The zygomatic bone forms the lateral orbital rim, while the frontal bone forms the superior orbital rim.

The superior rim is commonly indented by a small notch known as the supra orbital notch. This notch is invariably present at the junction of medial and lateral 1/3. The supra ortbital nerve and artery pass through this notch to reach the forehead.



Bones constituting orbit

The medial portion of the orbital rim is formed by the frontal process of maxilla and the maxillary portion of the frontal bones. A depression known as the lacrimal fossa is formed in the infero medial orbital rim. This fossa is formed by the maxillary and lacrimal bones. This lacrimal fossa is bounded by two projections of bones i.e. the anterior lacrimal crest of maxillary bone and the posterior lacrimal crest of lacrimal bone. This fossa houses the nasolacrimal sac. This fossa opens in to the nasolacrimal canal through which the naso lacrimal duct traverses.

The naso lacrimal duct is 3 – 4 mm in diameter, courses in an infero lateral and slightly posterior direction towards the inferior turbinate under which it opens into the inferior meatus. This duct is roughly 12mm long. All the walls of the lacrimal duct except its medial wall is formed by the maxillary bone. The medial wall is formed by the lateal nasal wall inferiorly and the descending process of lacrimal bone superiorly.

In the frontal process of maxilla just anterior to the lacrimal fossa a fine groove known as the sutura longitudinalis imperfecta of Weber. This suture runs parallel to the anterior lacrimal crest. Small branches of infraorbital artery pass through this groove to supply the nasal mucosa. The presence of these vessels should be anticipated in any lacrimal sac surgery to avoid unneccessary troublesome bleeding.

Medial wall of orbit:

Largest bony component of medial wall is the quadrangular shaped orbital plate of ethmoid bone. This bony component separates orbit from the nasal cavity. This plate of bone articulates superiorly with the medial edge of orbital plate of frontal bone. These articulating bony structures have two notches (anterior and posterior ethmoidal notches). These notches in these bones when combined forms the anterior and posterior ethmoidal canals. These canals transmit the ethmoidal branches of nasociliary nerve (branch of ophthalmic division of trigeminal nerve) and branches of opthalmic artery. These branches from opthalmic artery (anterior and posterior ethmoidal arteries) supply nasal

mucosa and duramater of frontal bone area. The cranial opening of these ethmoidal canals are related to the anterior and posterior limits of cribriform plate. The roof of the nasal cavity is partially formed by the cribriform plate of ethmoid. These cranial openings of ethmoidal canals divide anterior skull base into frontal, cribriform, and planum areas. Ethmoidal canals divide orbit into bulbar, retrobulbar and apical portions. This intricate knowledge of orbital anatomy helps during advanced endoscopic skull base surgical procedures[2] .

The medial wall of the orbit is formed from anterior to posterior by :

1. frontal process of maxilla

2. lacrimal bone

3. ethmoid bone

4. lesser wing of sphenoid bone

The thinnest portion of the medial wall is the lamina papyracea which separates the ethmoidal sinuses from the orbit. It is one of the components of ethmoid bone. Infections from ethmoidal sinus can easily breach this paper thin bone and affect the orbital contents. The medial wall of the orbit is thicker posterior where the sphenoid bone is present and anteriorly where the posterior lacrimal crest is present.

The fronto ethmoidal suture line marks the approximate level of ethmoidal sinus roof, hence any dissestion above this line may expose the cranial cavity. The anterior and posterior ethmoidal foramina through which branches of ophthalmic artery (anterior and posterior ethmoidal arteries) and branches of naso ciliary nerve passes are present in this suture. The anterior ethmoidal foramen is located at a distance of 24 mm from the anterior lacrimal crest, while the posterior ethmoidal foramen is located at a distance of 36 mm from the anterior lacrimal crest.

A vertical suture that runs between the anterior and posterior lacrimal crests is the anastomotic area between the maxillary and the lacrimal bone. If this suture is located more anteriorly it indicates a predominance of lacrimal bone, while a more posteriorly placed suture line indicates a predominance of maxillary bone in the anastomotic relationship. The lacrimal bone at the level of lacrimal fossa is pretty thin (106 micrometer). This bone can be easily penetrated during dacryocystorhinostomy surgery. If the maxillary component is predominant it becomes difficult to perform the osteotomy in this area to access the sac because the maxillary bone is pretty thick. Hence lacrimal bone predominance makes it easy to expose the sac during dacryocystorhinostomy [3] .

Applied anatomy of medial wall of orbit:

This wall is aligned parallel to the antero posterior axis and is very fragile because of its proximity to anterior ethmoidal air cells. Disruption of this wall due to trauma causes hypertelorism (Traumatic Hypertelorism). Lateral displacement of frontal process of maxilla will cause traumatic telecanthus because the medial palpebral ligament is attached here. Both hypertelorism and telecanthus can be caused due to trauma.

Contribution of ethmoid bone:

Ethmoid bone forms the medial boundary of orbit. It is separated from obital contents by a paper thin bone (Lamina papyracea). This bone can be breached due to diseases involving ethmoids or during nasal surgeries allowing infections to reach the orbital cavity. Inferiorly ethmoid bone articulates with the orbital plate of maxilla. Posteriorly the ethmoid bone articulates with the body of sphenoid

completing the medial bony wall of orbital cavity.

Sphenoid bone:

Sphenoid bone contributes to the formation of bony orbit by its greater and lesser wings. The lesser wings of sphenoid articulates with orbital plate of frontal bone to form the roof of orbit. The greater wings of sphenoid articulates laterally with the orbital plate of zygoma forming the lateral wall of bony orbit.

Lateral wall of orbit:

Understanding this wall of the orbit is vital from the surgeon's point of view. Two components are involved in the formation of this wall. The greater wing of sphenoid faces the orbit on its exocranial side and its endocranial surface forms the anterior limit of middle cranial fossa. The zygomatic bone on the contrary does not have cerebral surface / endocranial surface. It virtually faces the orbit while its opposite surface froms the anterior limit of infratemporal fossa. This anatomical relationship provides lateral access to the orbit without resorting to craniotomy. In the lateral orbital approach, the contents of the orbit can be reached just by displacing the temporal bone and performing zygomatic osteotomy.

The recurrent meningeal branch of middle meningeal artery may be seen coursing through a foramen in the suture line between the frontal and sphenoid bones. This artery forms a anastomosis between the external and internal carotid arterial systems. Roughly 4 – 5 mm behind the lateral orbital rim and 1 cm inferior to the frontozygomatic suture is the lateral tubercle of Whitnall. The following structures gets attached to this tubercle:

1. Lateral canthal tendon

2. Lateral rectus check ligament

3. Suspensory ligament of lower eyelid (Lockwoods ligament).

4. Orbital septum

5. Lacrimal gland fascia.

Lateral canthal tendon:

The pretarsal muscles join laterally to form the lateral canthal tendon. This tendon inserts into the periosteum of Whitnall's tubercle about 5 mm behind the infraorbital rim.

Lateral rectus check ligament:

This is a fibrous membrane arising from the lateral rectus muscle and gets attached to the zygomatic tubercle, posterior aspect of lateral palpebral ligament and the lateral conjunctival fornix.

Being most prone for injury this wall of the orbit happens to be the thickest. It is very strong at the orbital margin. Behind this thick portion of lateral wall comes the somewhat thinner portion, behind this thin portion the wall again becomes thick. Posterior most portion of this lateral wall is thin (about 1 mm) nearly translucent.

This wall is further weakend by the presence of superior orbital fissure between lateral and superior walls of orbit. The presence of inferior orbital fissure between lateral and inferior walls of orbit creates another area of weakness.

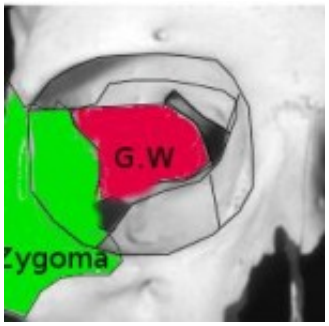


Diagram showing lateral wall of orbit: GW (Greater wing of sphenoid)

Superior orbital fissure:

This is also known as sphenoidal fissure because it lies between lesser and greater wings of sphenoid. This space is closed laterally by the frontal bone. This fissure lies between the lateral wall and roof of orbit. At its medial end it is slightly wider. At this point it lies below the optic foramen. This fissure gradually reduces in size as it reaches its lateral extremity. Superior orbital fissure hence should be considered to have a narrow lateral and a wide medial part. This fissure is about 22 mm long and is the largest communication between the orbit and the middle cranial fossa. Its tip is situated about 30-40 mm from the frontozygomatic suture line. Its medial end is separated from optic foramen by the posterior root of lesser wing of sphenoid. This portion of sphenoid bone has a small tubercle known as infraoptic tubercle. The annulus of Zinn from which all the intraocular muscles originate spans the superior orbital fissure between its medial wide and lateral narrow portions. Annulus of Zinn surrounds the optic nerve at its entrance into the orbit.

The following structures pass through the annulus of Zinn:

1. Superior division of 3rd nerve

2. Nasociliary nerve

3. Sympathetic root of cervical ganglion

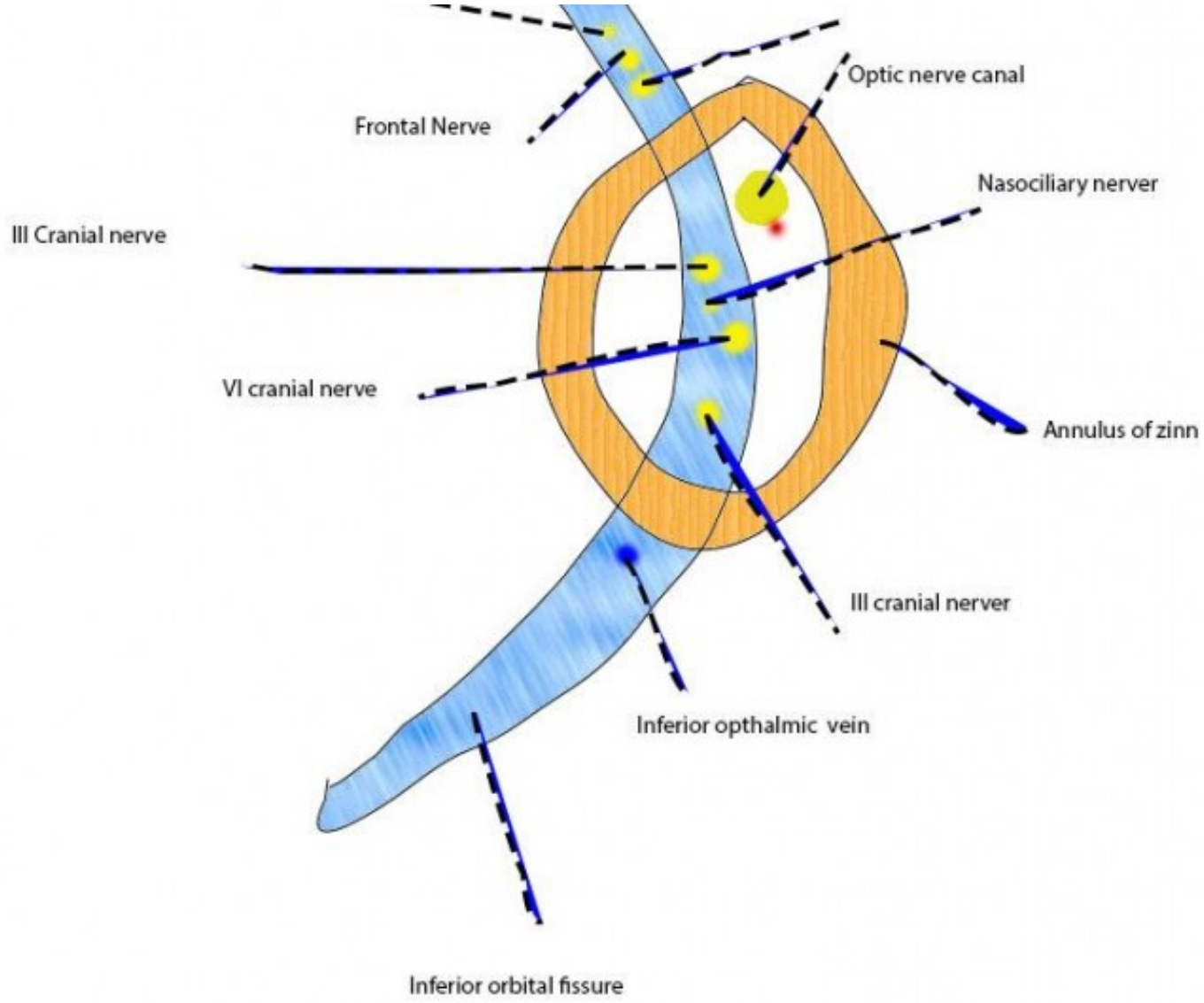
4. Inferior division of 3rd nerve

5. 6th nerve

6. Opthalmic vein (superior opthalmic)

This is the rough order of structures passing through the annulus from above downwards.

Inferior opthalmic vein passes below the annulus.



Lockwood's ligament:

This ligament acts as a hammock supporting the globe inferiorly. This is actually a dense condensation of connective tissue engulfing inferior rectus and inferior oblique muscles providing support to the undersurface of the globe. This ligament is attached to facial structures connected to the lower lid. Damage to Lockwood's ligament can cause lower eyelid ptosis which is seen in patients

undergoing total maxillectomy.

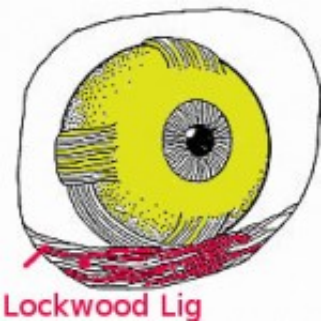


Figure showing Lockwood's ligament acting as a Hammock holding the globe

Orbital septum:

This is also known as palpebral ligament. This membranous sheet acts as the anterior boundary of the orbit [4] . It extends from the orbital rims to the eyelids. With age this septum may weaken causing prolapse of orbital fat forwards. Blepharoplasty is usually performed to correct this anamoly. Orbital septum helps in differentiating orbital cellulitis (behind the septum) and periorbital cellulitis (in front of the septum) [5] . This structure is usually penetrated by vessels and nerves that pass from the orbit to face and scalp.

The frontal process of zygomatic bone and the zygomatic process of frontal bone are thick and they protect the globe from lateral trauma. Just behind this facial buttress area the posterior zygomatic bone and the orbital plate of greater wing of sphenoid are thinner thus making the zygomatico sphenoid suture a convenient land mark for lateral orbitotomy. The zygomatico facial and zygomatico temporal nerves and vessels pass through the lateral wall of the orbit to reach the cheek and temporal regions. Posteriorly the lateral wall thickens and meets the temporal bone which forms the lateral wall of the cranial cavity. When lateral orbitotomy is being done only 12 – 13 mm separate the posterior aspect of lateral orbitotomy to that of the middle cranial fossa. This distance could still be shorter in females.

Foramen and fissures of orbit:

The following are the various foramina and fissures of orbit:

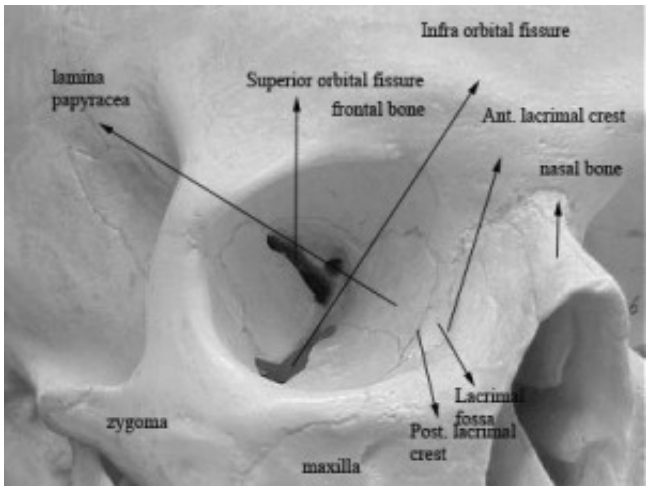
1. Superior orbital fissure (sphenoidal fissure)

2. Inferior orbital fissure (sphenomaxillary fissure)

3. Anterior and posterior ethmoidal canals

4. Optic canal/foramen

Orbit showing various components



Inferior orbital fissure:

Also known as spheno-maxillary fissure.

This same combination of zygomatic bone and greater wing of sphenoid forms the posterior border of infraorbital fissure. The anterior border is formed by orbital plate of maxilla and the postero medial part is formed by orbital process of palatine bone.

The inferior orbital fissure lies between the lateral orbital wall and the floor of the orbit. It is about 20 mm long. This is also known as sphenomaxillary fissure. It is bounded anteriorly by the maxilla and the orbital process of palatine bone, posteriorly by the lower margin of orbital surface of greater wing of sphenoid. This fissure is narrower at its center when compared to its extremities. The actual width of this fissure is dependent on the development of maxillary sinus. This fissure is somewhat wider in infants and children. This fissure lies near the openings of foramen rotundum and the sphenopalatine foramen.

The following structures pass through this fissure:

1. Maxillary division of trigeminal nerve

2. Zygomatic nerve

3. Branches from the sphenopalatine ganglion

4. Branches of inferior ophthalmic vein leading on to pterygoid plexus.

The maxillary division of trigeminal nerve and the terminal branch of internal maxillary artery enter the infra orbital groove and canal to become the infra orbital nerve and artery. These structures exit through the infra orbital foramen to supply the lower eye lid, cheek, upper lip and upper anterior gingiva. The orbit communicates with pterygopalatine fossa through the medial most portion of this fissure and through this into the nasal cavity. Laterally infraorbital fissure is in contact with temporal and infratemporal fossa. This lateral aspect of the fissure is filled with smooth muscle and fat tissue making it a suitable place for bone cuts.

In living persons this fissure is closed by periorbital tissue and Muller’s muscle.

Floor of the orbit:

This area is actually interesting because it can be involved in pure blow out fractures without involvment of zygoma. It is more or less triangular in shape with rounded corners. It is narrow

posteriorly. This is actually not horizontal, but slopes upwards and medially at an angle of 45°. It ends as the anterior margin of inferior orbital fissure. In this area this bone abruptly curves downwards towards the infratemporal fossa forming the posterior wall of maxilla.

Components of the floor of the orbit:

1. Orbital plate of maxilla (largest component)

2. Orbital plate of zygomatic bone (antero lateral part)

3. Orbital process of palatine bone (forms a small portion behind the maxilla)

The floor of the orbit is traversed by inferior orbital fissure. This fissure infact weakens the floor. Most of blowout fractures occur medial to this fissure. Fracture line can cause entrapment of infraorbital nerve leading on to anesthesia of cheek area of that side. Infraorbital canal formed from this fissure sinks anteriorly and opens into the infraorbital foramen.

The roof of the orbit slopes down medially. In fact this slope continues up to fronto ethmoidal suture to form the roof of the ethmoid sinus. This is otherwise known as fovea ethmoidalis.

The anatomical relationship between the anterior ethmoidal air cells and the lacrimal fossa should be borne in mind to avoid confusion between the ethmoid and nasal cavities during dacryocystorhinostomy surgery.

Ethmoidal foramen:

These foramina lie between the roof and medial wall of orbit. These foramina invariably lie within the frontoethmoidal suture line or in the frontal bone. These openings form canals known as anterior and posterior ethmoidal canals. These canals are formed by frontal bone to a great extent with minor contributions from ethmoids.

Anterior ethmoidal canal:

This canal is directed backwards and laterally. This foramen is located about 24 mm from the anterior lacrimal crest. The posterior border of this canal is not well defined and is continuous with a groove on the orbital plate of ethmoid. This canal opens into the anterior cranial fossa at the side of cribriform plate transmitting anterior ethmoidal nerve and artery.

Figure showing anterior ethmoidal artery

Posterior ethmoidal canal:

This canal lie posterior to anterior ethmoidal canal. This foramen is located 36 mm from the anterior lacrimal crest. It transmits posterior ethmoidal nerve and posterior ethmoidal artery.

Optic foramen:

Also known as optic canal. It begins from the middle cranial fossa and extends up to the apex of the orbit. This foramen is formed by two roots of the lesser wing of sphenoid. This foramen is directed laterally, forwards and downwards. This canal is funnel shaped, the mouth of the funnel is its anterior opening. This foramen is oval in shape with the vertical diameter being the greatest. Its intracranial opening is flattend above downwards, where as its middle portion is circular in nature. Its lateral border is well defined and is formed by the anterior border of the posterior root of lesser wing of sphenoid. Its medial border is less well defined. Optic canal is separated from the medial end of superior orbital fissure by a bar of bone. This bar of bone has a tubercle for the attachment of annulus tendinous.

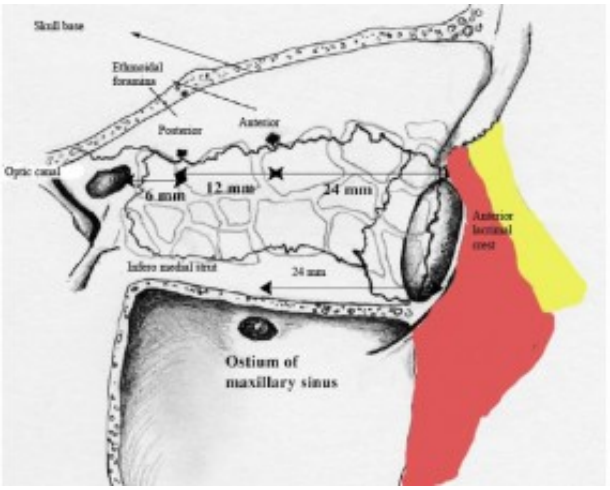


Diagram showing view of skull after removal of lateral wall

Optic nerve canal transmits:

1. Optic nerve

2. Coverings of optic nerve including duramater, arachnoidmater and piamater.

3. Opthalmic artery lies below and lateral to the nerve embedded in the dural sheath

Orbital index:

The width of the orbit is larger than that of its height.

Orbital index varies among various human races. Going by orbital index 3 types of orbits have been identified.

Orbital index= height of the orbit

_____ X 100

Width of the orbit

Megaseme: This is a rather large orbital index. Here the orbital index calculated using the formula above is more than 89. This orbit is the classic feature of yellow races.

Mesoseme: This is intermediate orbital index. Here the index ranges between 83-89. This is commonly seen in European whites.

Microseme: This is the smallest orbital index. The value here is less than 83. This orbit is typically seen in black races [6] . Orbital opening is rectangular.

Orbital margin:

This is made up of three bones.

Frontal

Zygomatic

Maxilla

Superior orbtial margin:

This is entirely formed by frontal bone. This portion of frontal bone is also known as orbital arch. This margin is sharp in lateral 2/3 and rounded in medial third. At the junction of these two portions about 25 mm from midline is situated the supraorbital notch. This notch transmits supraorbital vessels and nerves. This notch is converted into a foramen due to ossification of the ligament which lies inferior to this notch. This notch can easily be palpated in the living.

Arnold's notch:

This is rarely seen medial to supraorbital notch. This notch is also known as the Arnold's notch. This notch transmits the medial branches of supraorbital vessels and nerves.

Lateral orbtial margin:

This margin is the most exposed one and is the strongest of the orbital margins. It is formed by the zygomatic process of frontal bone and zygomatic bone. The lateral orbital rim is recessed to accomodate lacrimal gland. This recess may be involved in segmental frature in this region. The narrowest and weakes part of this rim is the frontozygomatic suture line. Separation of this suture line is a common feature of trauma in this region.

Inferior orbtial margin:

This margin is raised sligthly above the floor of the orbit. This margin is formed by zygomatic bone and maxilla in equal proportions. The infraorbital margin is clearly defined at its lateral margin and is easily palpable. Inner portion of the rim is rounded and is not easily palpable.

Medial margin:

This margin is formed by anterior lacrimal crest present on the frontal process of maxilla and the posterior lacrimal crest on the lacrimal bone. The medial margin is hence not a continuous ridge.

Age changes in orbit:

Anatomical changes involving orbit depends on the development of facial skeleton and the neighbouring paranasal sinuses.

At birth the orbital margins are sharp and completely ossified. This helps in protecting the eyes during the stressful event of parturition. At about the age of 7 the orbital margins but for the superior margin become fairly rounded and less sharp. At this age the superomedial and inferolateral angles are well

marked than other angles. This causes the orbit to be triangular.

Infant's orbit look more laterally than adults.

Orbital fissures are large in a child when compared with that of adults. This is because of the narrow orbtial surface of greater wing of sphenoid.

The orbital index is higher in a child when compared to adults. The vertical diameter is the same as that of horizontal diameter. As growth progresses the transverse diameter increases more than the vertical..

The interorbital distance is rather small in children. This mimiks squint.

The roof of the orbit is much larger than the floor at birth.

In infants optic canal is not a canal. It is just a foramen. As the infant grows this foramen elongates to become the optic canal.

The periorbita is thicker and stronger at birth than in adults.

Old age changes occuring in the orbit are actually due to bone absorption. The roof of the orbit in elderly person may actually contain holes which causes periorbita to come into direct contact with dura. Parts of lacrimal bone too can be absorbed due to ageing process.

Soft tissues of orbit:

Orbital septum is the anterior soft tissue boundary of the orbit. It acts as a physical barrier against pathogens. This is a thin multilayered fibrous tissue derived from the mesodermal layer of eyelid. This septum is covered anteriorly by the preseptal orbicularis oculi muscle.

Periorbita: is the periosteal lining of orbital walls. The periorbita is attached to the suture lines, fissures and foramina of the orbit. Posteriorly the periorbita is continuous with the optic nerve sheath.

Orbital fat: Adipose tissue present in the orbit has a cushioning effect on the contents of orbit.

The extra ocular muscles of orbit arise from the annulus of zinn and are responsible for the movement of the globe. These muscles are:

lateral and medial rectus

Superior and inferior rectus

Superior and inferior oblique

The four recti muscles arise from the annulus of zinn. The annulus of zinn actually has two tendons. The lower tendon of annulus of zinn is attached to the medial end of superior orbital fissure enclosing the optic foramen. This tendon gives origin to parts of medial and lateral recti. It also gives attachment of entire inferior rectus muscle. The upper tendon of the annulus of zinn also known as tendon of Lockwood arises from the body of sphenoid. This tendon gives origin to part of medial and lateral recti and all of the superior rectus muscle. The attachments of superior and medial recti muscles are close to the dural sheath of optic nerve. This fact explains the pain caused during extremes of eye movements in retrobulbar neuritis.

Medial rectus:

This is the largest of the ocular muscles. It is also stronger than the lateral rectus. From its origin from

the annulus of Zinn it inserts into the globe medially 5.5 mm from the limbus. Its blood supply is derived from the inferior muscular branch of opthalmic artery and anterior ciliary arteries. It derives its motor innervation from the third cranial nerve on its lateral surface at the junction of middle and posterior thirds. It is a pure adductor.

Inferior rectus:

This is the shortest of all recti muscles. From its origin in the annulus of zinn close to optic foramen it inserts into the globe inferiorly 6.5 mm from the limbus. It is also attached to the lower eye lid via its facial expansion. It derives its blood supply from the inferior muscular branch of ophthalmic artery, infraorbital artery and anterior ciliary vessels. It derives its motor innervation from the inferior division of third nerve on its upper aspect at the junction of its middle and posterior thirds. It moves the eye downwards and medially / rotates it laterally (extorsion). It can also depress the lower eye lid by its facial sling which inserts into it. Its principal action is depression of out turned eye. Infact it is the only depressor of the abducted eye.

Lateral rectus:

From its origin from the annulus of zinn it is inserted laterally into the globe about 6.9 mm from the limbus. It receives blood supply from lacrimal artery. It is the only ocular muscle with single source of blood supply. It is innervated by the 6th cranial nerve in its medial aspect. It is a pure abductor making the eye to look directly laterally.

Superior rectus muscle:

Arising from the superior portion of annulus of zinn it is inserted in to the bulb superiorly about 7.7 mm from the limbus. It receives its blood supply from the superior muscular branch of ophthalmic artery and anterior ciliary arteries. It is innervated by superior division of oculomotor nerve. This nerve enters the undersurface of the muscle at the junction of middle and posterior thirds. It helps in upwards and medial rotation of the eye and is also capable of intorting the eye ball.

Superior oblique muscle:

This is the longest and thinnest of ocular muscles. It arises medial to the optic foramen and gets inserted into the trochlea on the orbital rim (on the anterosuperior portion of the medial wall of orbit). Its tendon gets inserted on to the temporal aspect of the eye behind the equator. The superior muscular branch of opthalmic artery and ciliary arteries supply this muscle. It moves the eye downwards and laterally. It is the only muscle that can depress the eye in adducted position. It is supplied by the 4th nerve.

Inferior oblique:

This is the only extrinsic muscle to take origin from the front of the orbit. This muscle arises from the orbital floor in a depression near the orbital rim. Some of its fibres may also arise from the fascia covering lacrimal sac. It is inserted into the posterior inferior temporal quadrant at the level of macula. It derives its blood supply from the inferior branch of ophthalmic artery and infra orbital artery. It is innervated by the inferior division of oculomotor nerve. This nerve enters the muscle from its upper surface. This muscle helps the eye to look upwards and laterally and in extorsion of orbit. This is the only muscle that elevates the eye in the adducted position.

Levator palpebrae superioris:

This striated muscle elevates the eyelid. This muscle arises from the under surface of lesser wing of

sphenoid just above and in front of optic foramen, and usually it is blended with the origin of superior rectus muscle. From this attachment this ribbon like muscle passes forwards below the roof on top of the superior rectus muscle. It gets inserted into the skin of the upper eyelid, and upper tarsal plate. It receives its nerve supply from the superior divison of 3rd cranial nerve. This muscle by its elevating action raises the upper eyelid, thus uncovers the cornea and portions of sclera. The action of this muscle is antogonized by orbicularis oculi muscle inneravated by facial nerve.

Muller's muscle:

This smooth muscle acts as an eyelid elevator. It arises from the inferior aspect of levator palpebrae. This muscle is inserted into the upper edge of tarsal plate. It is innervated by sympathetic fibers. The action of this muscle accounts for the presence of upper lid elevation in patients with 3rd cranial nerve palsy.

The lacrimal system:

The main lacrimal gland is located in the supero temporal portion of orbit. It lies in the shallow lacrimal fossa of the frontal bone. The gland is composed of numerous secretory units known as acini which progressively drain in to small and larger ducts. The gland measures 20 mm by 12 mm. A fibrous band incompletely devides the lacrimal gland into two lobes i.e. posterior larger orbital lobe and a smaller anterior palpebral lobe. 2 – 6 ducts from the orbital lobe pass through the palpebral lobe joining with the ducts from the palpebral lobe to form 6 – 12 tubules to empty into the superio lateral conjunctiva. Hence damage to the palpebral lobe may block drainage from the entire gland. About 20 – 40 accessory lacrimal glands of Krause are located in the superior conjuctival fornix, about half this number is located over the lower fornix.

The lacrimal gland is innervated by branches from 5th and 7th cranial nerves, sympathetic supply to lacrimal gland is via the nerves from the superior cervical ganglion. The parasympathetic fibers are supplied via the 6th nerve. Sensory supply is via the branches of trigeminal nerve.

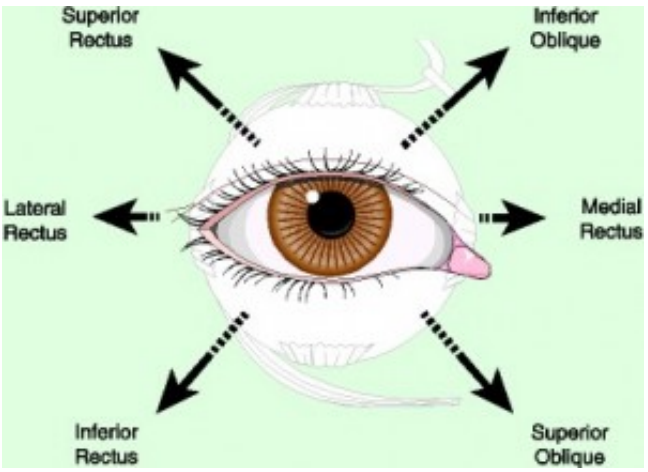


Diagram illustrating roles played by various muscles in ocular movement

The lacrimal excretory system begins at a 0.3 mm at the medial end of each eyelids known as the punctum. These puncta are directed posteriorly. The punctal opening widens into ampulla, which is perpendicular to the eye lid margin. The ampulla makes a sharp turn to drain into the canaliculi. The canaliculi measures 0.5 – 1mm in diameter and courses parallel to the lid margins. The superior canaliculus is 8 mm long and the inferior canaliculus is 10 mm long. In majority of individuals the superior and inferior canaliculi merge into a common canaliculi before draining into naso lacrimal sac. The opening of common canaliculi into the naso lacrimal sac is known as the common internal

punctum. There is a valve at the junction of common canaliculus and lacrimal sac at the common internal punctum level. This is known as the Rosenmuller valve. Another valve known as the valve of Hasner is found at the lower end of the naso lacrimal duct at the level of inferior meatus of nose.

If this Hasner's valve is imperforate in new born infants it causes congenital naso lacrimal obstruction.

The lacrimal sac resides in the lacrimal fossa. It measures about 12 – 15 mm vertically, and 4 – 8 mm antero posteriorly.

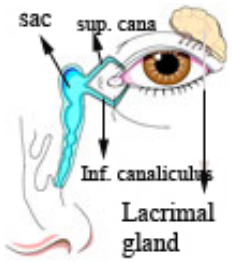


Diagram showing lacrimal apparatus

Location of lacrimal sac:

A good intranasal landmark for the location of lacrimal sac is the anterior portion of middle turbinate, the sac lies just lateral to it. The lacrimal fossa is bounded by the anterior lacrimal crest, which consists of the frontal process of the maxillary bone. The posterior lacrimal crest is made up of the lacrimal bone itself.

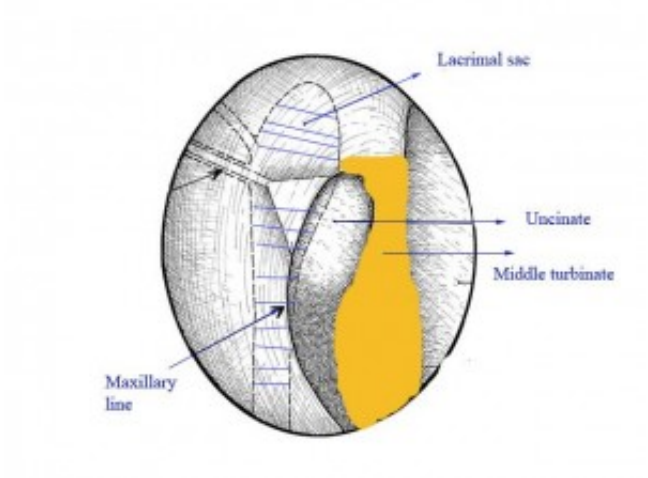


Figure showing intranasal landmark of lacrimal sac

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March 2012

Atrophic rhinitis a review

Atrophic rhinitis

A review

March 3, 2012 · Rhinology

Authors

Balasubramanian Thiagarajan

Abstract

Atrophic rhinitis is a chronic embarrassing debilitating disease involving nasal cavity and paranasal sinuses. It is characterized by foul smelling crusts (greenish) inside the nasal cavity. Nasal mucosa tends to bleed when these crusts are removed. Nasal cavity appears to be excessively roomy. Foul stench emanates from the patient. Patient is mercifully unaware of this stench because of the presence of anosmia. This article discusses the various features of this disease and the various management modalities available.

Atrophic rhinitis a review

Introduction:

Atrophic rhinitis is defined as a chronic nasal disease characterised by progressive atrophy of the nasal mucosa along with the underlying bones of turbinates. There is also associated presence of viscid secretion which rapidly dries up forming foul smelling crusts. This fetid odor [1]is also known as ozaena. The nasal cavity is also abnormally patent. The patient is fortunately unaware of the stench emitting from the nose as this disorder is associated with merciful anosmia[2]. This disease is rather rare in developed countries, but are rather common in developing countries [3]. Now a days it is more common as a sequelae of medical interventions. Overzealous turbinate surgery has been implicated as a probable iatrogenic cause.[4]

Synonyms:

The following are the various terminologies used to indicate the same condition:

1. Rhinitis sicca
2. Dry rhinitis
3. Ozena
4. Open nose syndrome
5. Empty nose syndrome

History:

It was Spenser Watson of London who first coined the term Ozena to describe this condition. He was the first to describe clinical features of this disease. He classified this disease into mild, moderate and severe varieties. Mild variety of atrophic rhinitis according to Watson ischaracterizedby heavy crusting. These crusts can easily be removed by nasal douching. Moderate variety features anosmia and stench emanating from the nasal cavity. Severe variety is invariably caused by syphilis. Bone destruction and cosmetic deformities of nose is rather common in this variety.[3]In 1876 Dr Bernhard Fraenkel first described the classic triad of symptoms which was virtually diagnostic of this disorder.

This triad is still known as Fraenkel's triad in his honor include: Fetor, Crusting and Atrophy of nasal structures. Frank Bosworth in 1881 noted that '*Breath from these patients were not only unpleasant but virtually unbearable. The sufferer perse was mercifully unaware because of the presence of anosmia*."

Etiology:

The etiology of this problem still remains obscure. Numerous pathogens[5] have been associated with this condition, the most important of them are:

1. Coccobacillus

2. Bacillus mucosus

3. Coccobacillus foetidus ozaenae

4. Diptheroid bacilli

5. Klebsiella ozaenae.

These organisms despite being isolated from the nose of diseased patients have not categorically been proved as the cause for the same.

Other predisposing factors include:

1. Chronic sinusitis -SSali [6]considered atrophic rhinitis to be infective in nature. He reported atrophic rhinitis in 7 children of a family after a child with atrophic rhinitis spent a night in their house. Common organism isolated from nasal cavities of these children was Klebsiella ozenae

2. Excessive surgical destruction of the nasal mucosa and turbiantes

3. Nutritional deficiencies: Bernat [7]in 1965 demonstrated that 50% of patients with atrophic rhinitisbenefitedwith iron therapy. Han-sen[8] demonstrated symptomatic improvement in majority of this patients with atrophic rhinitis when treated with vitamin A.

4. Syphilis.

5. Endocrine imbalances (Disease is known to worsen with pregnancy / menstruation)

6. Heredity: This was first reported by Barton and Sibert[9](Autosomal dominent pattern of inheritance identified).

7. Autoimmune disease

8. Developmental: Hagrass[10] reported shortened Antero Posterior nasal lengths and poor maxillary antral pneumatization in patients with atrophic rhinitis.

9. Vascular: Excess sympathetic activity was observed in these patients by Ruskin

Age of onset:

Usually atrophic rhinitis commences at puberty.[2]

Sex predilection:

Females are more commonly affected than males

Classification of Atrophic rhinitis:

Primary atrophic rhinitis: This classic form of atrophic rhinitis is supposed to arise de novo. This isin facta diagnosis of exclusion. This type of atrophic rhinitis is common in China, India and Middle East.

In almost all these patients Klebsiella ozenae has been isolated.

Secondary atrophic rhinitis: This is the most common form of atrophic rhinitis seen in developed countries. In this type the predisposing cause is clearly evident. Common causes of secondary atrophic rhinitis include:

1. Surgical procedures involving nose and paranasal sinuses – They include turbinectomies, sinus surgeries, maxillectomy etc.

2. Irradiation

3. Trauma

4. Granulomatous diseases: Include Sarcoidosis, Leprosy and Rhinoscleroma

5. Infections: This includes tuberculosis and syphilis

Clinical features:

The presenting symptoms are commonly nasal obstruction and epistaxis. Anosmia i.e. merciful may be present making the patient unaware of the smell emanating from the nose. These patients may also have pharyngitis sicca. Choking attacks may also be seen due to slippage of detached crusts from the nasopharynx into the oropharynx. These patients also appear to be dejected and depressed psychologically.

Clinical examination of these patients show that their nasal cavities filled with foul smelling greenish, yellow or black crusts, the nasal cavity appear to be enormously roomy. When these crusts are removed bleeding starts to occur.

Why nasal obstruction even in the presence of roomy nasal cavity?[2]

This interesting question must be answered. The nasal cavity is filled with sensory nerve endings close to the nasal valve area. These receptors sense the flow of air through this area thus giving a sense of freeness in the nasal cavity. These nerve endings are destroyed in patients with atrophic rhinitis thus depriving the patient of this sensation. In the absence of these sensation the nose feels blocked.

Radiologic features of atrophic rhinitis:

Radiologic features are similar for both types of atrophic rhinitis. Plain xrays show lateral bowing of nasal walls, thin or absent turbinates and hypoplastic maxillary sinuses.

CT scan findings:

1. Mucoperiosteal thickening of paranasal sinuses

2. Loss of definition of osteomeatal complex due to resorption of ethmoidal bulla and uncinate process

3. Hypoplastic maxillary sinuses

4. Enlargement of nasal cavity with erosion of the lateral nasal wall

5. Atrophy of inferior and middle turbinates

Fig. 1: CT scan nose and sinuses in a patient with atrophic rhinitis

Histopathological features:

1. Metaplasia of ciliated columnar nasal epithelium into squamous epithelium.

2. There is a decrease in the number and size of compound alveolar glands

3. Dilated capillaries are also seen

Pathologically atrophic rhinitis has been divided into two types:

Type I: is characterised by the presence of endarteritis and periarteritis of the terminal arterioles. This could be caused by chronic infections. These patients benefit from the vasodilator effects of oestrogen therapy.

Type II: is characterised by vasodilatation of the capillaries, these patients may worsen with estrogen therapy. The endothelial cells lining the dilated capillaries have been demonstrated to contain more cytoplasm than those of normal capillaries and they also showed a positive reaction for alkaline phosphatase suggesting the presence of active bone resorption. It has also been demonstrated that a majority of patients with atrophic rhinitis belong to type I category.

Management:

Conservative:

Nasal douching – The patient must be asked to douche the nose atleast twice a day with a solution prepared with:

Sodium bicarbonate – 28.4 g

Sodium diborate – 28.4 g

Sodium chloride – 56.7 g

mixed in 280 ml of luke warm water.

The crusts may be removed by forceps or suction. 25% glucose in glycerin drops can be applied to the nose thus inhibiting the growth of proteolytic organism.

In patients with histological type I atrophic rhinitis oestradiol in arachis oil 10,000 units/ml can be used as nasal drops.

Kemecetine antiozaena solution – is prepared with chloramphenicol 90mg, oestradiol dipropionate 0.64mg, vitamin D2 900 IU and propylene glycol in 1 ml of saline.

Potassium iodide can be prescribed orally to the patient in an attempt to increase the nasal secretion.

Systemic use of placental extracts have been attempted with varying degrees of success.

Surgical management:

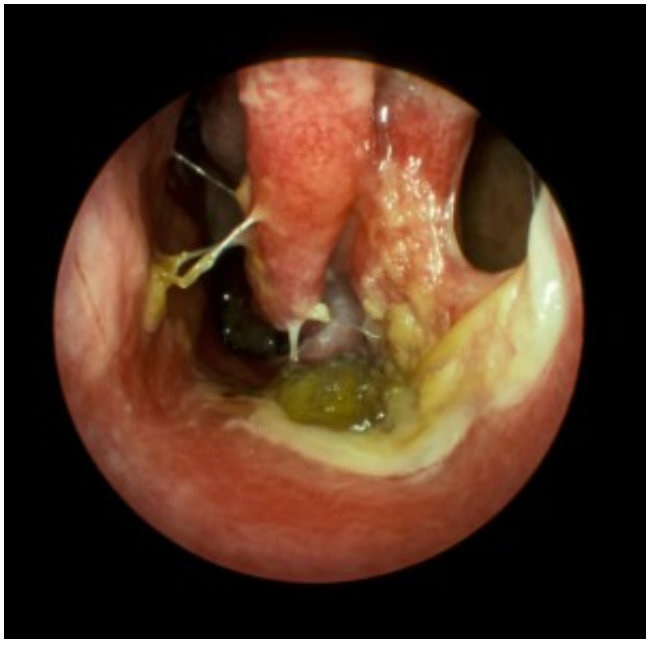
1. Submucous injections of paraffin, and operations aimed at displacing the lateral nasal wall medially. This surgical procedure is known as Lautenslauger's operation.

2. Recently teflon strips, and autogenous cartilages have been inserted along the floor and lateral nasal wall after elevation of flaps.

3. Wilson's operation – Submucosal injection of 50% Teflon in glycerin paste.

4. Repeated stellate ganglion blocks have also been employed with some success

5. Young's operation[11] – This surgery aims at closure of one or both nasal cavities by plastic surgery. Young's method is to raise folds of skin inside the nostril and suturing these folds together thus closing the nasal cavities. After a period of 6 to 9 months when these flaps are opened up the mucosa of the nasal cavities have found to be healed. This can be verified by postnasal examination before revision surgery is performed. Modifications of this procedure has been suggested (modified Young's operation) where a 3mm hole is left while closing the flaps in the nasal vestibule. This enables the patient to breath through the nasal cavities. It is better if these surgical procedures are done in a staged manner, while waiting for one nose to heal before attempting on the other side.



Atrophic rhinitis endoscopic view

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May 2012

Blow out fracture orbit Endoscopic reduction A Novel Management Modality

Blow out fracture orbit Endoscopic reduction

A Novel Management Modality

May 4, 2012 · Rhinology

Authors

Balasubramanian Thiagarajan

Abstract

Blow out fracture of orbit involves fracture of orbital floor without fracture of infraorbital rim. This injury is common from frontal blow to orbit. Frontal blow to orbit causes increased intraorbital tension causing fracture of floor of the orbit (weak point) with prolapse of orbital content into the maxillary sinus cavity. This causes enophthalmos and diplopia. Infraorbital rim is not involved in pure blow out fracture, it is also involved then it should be considered as an impure blow out fracture 3. Entrapment of inferior rectus muscle between the fracture fragments will cause diplopia in these patients. This article discusses a novel endoscopic internal reduction of fractured fragments. Main advantage of endoscopic approach is the lack of facial skin incision. It is cosmetically acceptable.

Blow out fracture orbit Endoscopic reduction a novel management modality

Introduction:

Orbital floor fractures were first described by MacKenzie in Paris in 1884 [1]. Smith was the first to describe entrapment of inferior rectus between the fracture fragments. He was also the first to coin the term “Blow out fracture” [2]. Blow out fracture causes an increase in the intraorbital volume, this causes enopthalmos. Entrapment of inferior rectus muscle causes diplopia. These patients usually report to an opthalmologist since orbital signs and symptoms are predominant. Shere etal in their study conclude that nearly 14% of blow out fractures are caused by contact sports in a military population [4].

Case Report:

30 years old male patient came with complaints of:

Clinical photograph of a patient with blow out fracture orbit showing orbital swelling

1. Swelling right eye – 1 day duration
2. Double vision – 1 day duration

3. Bleeding from right nose – 1 day duration

History of injury on being struck by a cricket ball +

He gave no history of loss of consiousness.

On examination:

Swelling over upper and lower eyelids on the right side +

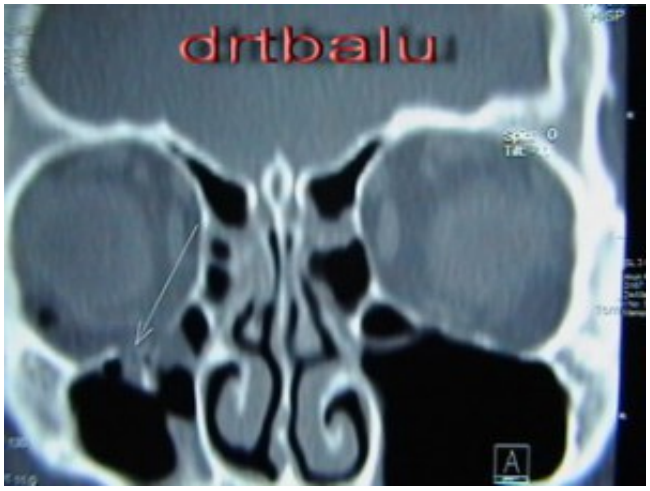
Enopthalmos right eye +

Ocular movements restricted on right gaze

Diplopia +

Forced duction test +

CT scan nose and paranasal sinuses:



Coronal CT plain of nose and sinuses showing blow out fracture right orbit (classic tear drop sign)

Showed evidence of blow out fracture right orbit. Tear drop sign could be seen.

Management:

Reduction was performed via Caldwel Luc approach under endoscopic guidance. 4 mm 30 degree nasal endoscope was used for this purpose. Trap door fractures can usually be reduced without resorting to prosthesis. Since this patient had a trap door fracture it could be easily reduced under endoscopic guidance. The reduced fracture fragment was stabilized by inflating the balloon of foley's catheter introduced into the maxillary sinus via inferior meatal antrostomy. Foley's catheter is left in place for a period of 2 weeks for union to occur.

Picture showing foley's catheter being introduced into the maxillary antrum via inferior meatal antrostomy

Picture showing inflated foleys catheter inside the maxillary antrum

Discussion:

Orbital blow out fracture is commonly caused by blunt trauma to the orbit. This is commonly seen in persons involved in contact sports like boxing, foot ball, rugby etc [5].

Two theories attempt to explain this injury phenomenon:

1. Buckling theory

2. Hydraulic theory

Buckling theory:

This theory proposed that if a force strikes at any part of the orbital rim, these forces gets transeferred to the paper thin weak walls of the orbit (i.e. floor and medial wall) via rippling effect causing them to distort and eventually to fracture. This mechanism was first described by Lefort 3.

Hydraulic theory [6]:

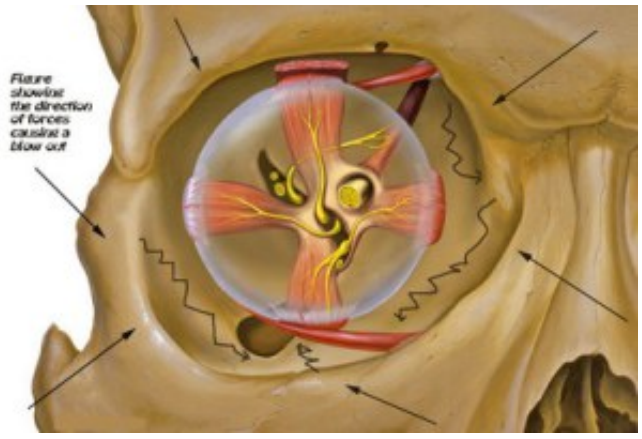
This theory was proposed by Pfeiffer in 1943. This theory believes that for blow out fracture to occur the blow should be received by the eye ball and the force should be transmitted to the walls of the orbit via hydraulic effect. So according to this theory for blow out fracture to occur the eye ball should sustain direct blow pushing it into the orbit.

Water House [7]in 1999 did a detailed study of these two mechanisms by applying force to the cadaveric orbit. He infact used fresh unfixed cadavers for the investigation. He described two types of

fractures:

Type I: A small fracture confined to the floor of the orbit (actually mid medial floor) with herniation of orbital contents in to the maxillary sinus. This fracture was produced when force was applied directly to the globe (Hydraulic theory).

Type II: A large fracture involving the floor and medial wall with herniation of orbital contents. This type of fracture was caused by force applied to the orbital rim (Buckling theory).



Diagrammatic representation of Buckling theory

Initial signs and symptoms of blow out fracture include:

1. Immediate swelling of the eye
2. Tenderness over involved orbit
3. Pain and difficulty with eye movements
4. Double vision
5. Enopthalmos
6. Numbness / tingling over lower eyelid, nose, upper lip[8]

Complications of blow out fracture:

1. Herniation of orbital fat into maxillary sinus[9]
2. Orbital emphysema[10]
3. Bleeding into maxillary sinus
4. Entrapment / rupture of ocular muscles
5. Ischaemic muscle contractures[11]
6. Cellulitis
7. Diplopia

Timing for surgical intervention:

This is highly controversial. Some of the authors prefer a waiting period of atleast 2 weeks for the oedema to resolve before proceeding with surgical reduction of the fracture. Early intervention is indicated only in white eyed blow out fracture which is common in children. In children the bones are flexible and does not break easily but bends. Significant amounts of orbital tissue may get entrapped in between the fractured fragments causing a compromise in their blood supply. This condition is

known as the white eyed blow out fracture. These patients should under go immediate reduction. Surgery is indicated if the eye has recessed by more than 2 mm into the ortbit, ocular movements restricted, persistence of diplopia.

Advantages of endoscopic approach:[12]

1. Accurate fracture visualization

2. Incisions are small

3. Facial incisions can be avoided

4. Minimal soft tissue dissection

5. Hospital stay minimized

6. Cosmetically acceptable[3]

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August 2012

Choanal atresia

Choanal atresia

August 30, 2012 · *Rhinology*

Authors

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Abstract: Choana is also known as posterior nasal aperture. Nasal airway continues with that of posterior nares. Air from nasal cavity finds its way into the lungs via the choanal apertures. In some children the choana may be congenitally closed. This causes either total (bilateral choanal atresia) or partial (unilateral choanal atreisa) nasal obstruction. Child being obligate nasal breathers, find it rather difficult to breathe when there is bilateral choanal atresia. This is more so during the first 6 weeks of life. Hence bilateral choanal atresia should be considered as an emergency in paediatric age group. This article attempts to discuss the etiopathology and management of this condition.

Introduction:

Choanal atresia is actually a developmental failure of the nasal cavity to communicate with nasopharynx.. This condition is rather rare occurring in about 1 in 10000 live births. This condition is more common in female children the ratio being 2:1 [1]. About 50% of of these patients have other associated congenital anamolies. The most commonly associated congenital anomaly is CHARGE Syndrome.

CHARGE Syndrome include [2]:

C- Coloboma

H- Heart disease

A- Choanal atresia

R- Mental and growth retardation

G- Genital hypoplasia

E- Ear deformities

Other anomalies associated with choanal atresia include [3]:

1. Polydactyly

2. Nasal / auricular deformities

3. Palatal deformities

4. Down’s syndrome

5. DiGeorge syndrome

6. Meningocele

7. Menigoencephalocele

8. Treacher Collin's syndrome

9.Mid face hypoplasia

Types of choanal atresia:

1. Unilateral / Bilateral : Unilateral choanal atresia is an incidental finding and is verycommon (about 70%) [4]. High degree of suspicion is necessary to identify this condition. An

infant with unilateral choanal atresia will have problems suckling milk from the breast opposite to the side of atresia.

2. Bony atresia – 90%, Membranous atresia – 6%, Combined atreisa 4% [5].

Anatomic deformitites associated with choanal atresia include [6]:

1. The bony atretic plate is situated infront of the posterior bony septum

2. Nasal cavity is narrow in these patients

3. Lateral pterygoid plates are found to be thickened compromising the nasal airway

4. Medially vomer is thickened

5. Inbetween lateral pterygoid and vomer is the membranous plate

6. High arched palate is common in these patients

History of choanal atresia:

This condition was first described by Johann George Roederer in 1755. This was later termed as an anatomical abnormality of palatine bone by Adolf Otto in 1854 [7].

Carl Emmert has been successfully credited with the first choanal atresia repair in 1854 [7].

Embryology of choanal atresia:

Development of face and cranial structures occur during the first 12 weeks of gestation. The development of choanae takes place between the 4th and 11th weeks of gestation [8]. Cranial

structures develop from neural crest cell migration. Development of nose begins during the 4th week of gestation. This is indicated by the formation of nasal pits. During the 5th week of gestation the nasal pits begin to fold inwards intothe mesenchyme forming nasal sacs. These primitive nasal sacs are separated from oral cavity by oronasal membranes. During the 8th week of gestation this oronasal membrane ruptures creating nasal cavity and a primitive choana located at the junction of nasal cavities and nasopharynx. During this phase of development there is gradual proliferation of neural crest cells. These cells contribute to the formation of skull base and nasal vaults. During the 10th week of gestation the nasal septum and developing palate fuse. The

primitive choanae gets pushed posteriorly. This choanae which forms during the 10th week of gestation is known as "Secondary choanae". In normal foetus these secondary choanae are patent

for a functioning airway between the anterior nasal cavity and nasopharynx [9].

Theories of development of choanal atresia [10]:

Four theories for the development of choanal atresia:

1. Persistence of a buccopharyngeal membrane from the foregut.

2. Persistence of the nasobuccal membrane of Hochstetter – most commonly accepted theory.

3. The abnormal persistence or location of mesodermal adhesions in the choanal region.

4. A misdirection of mesodermal flow secondary to local genetic factors better explains the popular theory of persistent nasobuccal membrane

Other theories that are not so widely accepted include:

1. Resorption of the floor of secondary nasal fossa

2. Incomplete dorsal extension of nasal cavity

3. Migration of dorsal part of fronto nasal process to fuse with the palatal shelves

Studies have revealed that cranio facial anamolies with mesenchymal damage and cell disruption were found in mothers who ingested high doses of vitamin A during their period of pregnancy.

This has been attributed to disturbances in migration pattern of neural crest cells, which is also followed by disturbances in mesoderm development in the cranio facial area [11].

In patients with choanal atresia the atretic plate has the following boundaries:

Superior – under surface of body of sphenoid

Lateral – medial pterygoid lamina

Medial – vomer

Inferior – horizontal plate of palatine bone

This anatomical knowledge of atrophic plate is highly valuable while performing surgery on these patients.

Clinical features:

Bilateral choanal atresia is considered to be a neonatal emergency. These infants present with asphyxia neonatorum. Bilateral choanal atresia is commonly associated with:

1. Nursing difficulties – Sucking difficulties

2. Respiratory distress – Cyclic. When the infant falls asleep it becomes breathless as the nose is blocked

3. Respiratory infections – can occur due to aspiration

4. Recurrent nasal allergies

5. Cyanosis which gets better when the child cries.

6. Cry is not normal (Rhinolalia clausa)

7. Bilateral choanal atresia is also commonly associated with other birth defects like orofacial defects, cardiac defects and limb defects [12].

Teratogenic syndromes causing bilateral choanal atresia include:

1. Methimazole embryopathy [13]

2. Carbimazole embryopathy [14]

Detailed drug intake history is a must in diagnosing embryopathies.

Unilateral choanal atresia is commonly missed. These infants find difficulty in sucking milk frombreast opposite to the side of choanal atresia. These children have unilateral nasal obstruction with nasal discharge. A strong degree of suspicion is a must to identify this condition.

Tests to identify choanal atresia:

1. Attempting to pass 6-8 sized French plastic catheter through the nose. If there is no atresia the catheter will effortlessly pass through the nasal cavity into the nasopharynx. If there is choanal atresia then a typical solid feeling would be encountered at about 3-3.5 cms from the alar rim. If obstruction is encountered within 1-2 cms from the anterior nares, then it could be caused due to traumatic deflection of nasal septum due to trauma.

2. When a wisp of cotton is placed closed to the nasal opening then it would move in the presence of air flow.

3. When methylene dye is instilled in to the anterior nasal cavity it can be seen passing through the nasopharynx. Obstruction due to choanal atresia will prevent flow of methylene dye into the nasopharynx.

CT scan imaging is virtually diagnostic:

It has the unique advantage of differentiating membranous choanal atresia from bony ones. In patients with combined atresia it will also reveal the contribution of these two elements to the atretic plate. Actual structures involved in the atretic plate would be clearly seen. In all these patients

vomer appears to be thickened, the lateral nasal wall bends medially to fuse with vomer thereby obstructing the nasal cavity.

Management:

In bilateral choanal atresia securing the air way takes the top priority. An oral airway can be introduced to tide over the immediate crisis.

Role of intraoral nipple:

A nipple with a large opening (McGovern) Nipple [15]can be introduced into the oral cavity of the infant to tide over the crisis. This provides adequate airway to the infant. A small infant feeding tube can be passed through another small opening present in the nipple or alongside the nipple for

gavage feeding. This helps to buy time till the child has gained adequate weight to withstand corrective surgery.

Tracheostomy:

This should be considered only on rarest of rare occasions when the patient is not able to adequately maintain oral airway.

Timing of repair in unilateral choanal atresia:

Choanal atresia repair in unilateral atresia is delayed till the child reaches its first birthday. This allows

the surgery site to enlarge thereby reducing the risk of post op stenosis. Bleeding is also reduced if surgery is delayed. Older infants tolerate stenting better than young ones.

Trans nasal approach:

Transnasal approach: (using endoscopes): The surgery is performed under general anesthesia. A self retaining nasal speculum is used to expose the nasal cavity and the atretic plate. If the atresia is membranous in nature a simple perforation of the same under endoscopic guidance would suffice.

The nasal cavity is decongested using 4% xylocaine with adrenaline in the concentration of 1 in 10,000 concentration. Under endoscopic guidance a mucosal incision is made and the mucosal flaps are elevated exposing the posterior vomer and lateral pterygoid lamina. A diamond burr on an angled hand piece is used to drill the atretic bony plate. It is perforated at the junction of the hard palate and the vomer. Incidentally this is the thinnest part of the atretic plate. This procedure was first described by Stankiewicz. To improve visualisation the inferior turbinate can be out fractured

or even be trimmed. After drilling care is taken to preserve the mucosal flaps. A silastic stent is placed into each nostril passing through the drilled neo choana. This helps in reducing the incidence of restenosis. Stent is kept in place for atleast 6 weeks 19.

Opening made should be large enough to allow smooth passage of suction catheter. 3-4 size Endotracheal tube can be used as stent to prevent restenosis. The size should be choosen carefully in such a way that it should be adequate to prevent restensosis and inadequate to cause nasal

regurgitation.

Caution:

While performing this procedure caution must be taken not to injure the sphenopalatine vessels behind the middle turbinate.

Advantages of this procedure:

1. This process is faster and easier

2. Blood loss is minimal

3. Can be performed in children of all ages who do not have associated external nasal deformities

4. Child can be immediately breast fed

5. Child can be discharged on the 3rd day itself

Disadvantages:

1. Vision is highly limited especially in the new born

2.Inability to adequately remove enough of the posterior vomerine septal bone and prevent restenosis

3. Longer stenting time

4. Endoscopes do not offer binocular vision

5. Cannot be done safely and with good results on patients with multiple nasal and nasopharyngeal

anomalies.

First transnasal repair of choanal atresia was performed by Dehaen[18] in 1985. He used microscope for magnification and visulization of the atretic plate.

Endoscopic transnasal approach[16] is facilitated by advances in instrumentation, anesthesia, imaging etc. CT imaging reveals the amount of contribution to the stenotic segment by the lateral nasal wall. It is prudent to avoid drilling too much into the lateral nasal wall as it could damage the

sphenopalatine vessels[17]. Use of powered instrumentation like soft tissue shavers and drills have made this procedure a lot safer.

Use of Mitomycin C[19] to prevent restenosis:

In addition to routine stenting restenosis can be prevented by topical application of Mitomycin -C, which is an antimetabolite known to inhibit fibroblast formation.

Complications of use of stents following transnasal repair of choanal atresia:

1. Formation of granulation tissue

2. Crust formation

3. Septal perforation

4. Persistent nasal discharge

Transpalatal approach:

This approach is more suitable for bilateral choanal atresia. Under general anesthesia the palate is exposed with a mouth gag. Palatal mucosa is infiltrated with 2% xylocaine with 1 in 100000 adrenaline. This infiltration serves the dual purpose of helping in flap elevation and providing much needed hemostasis during the entire surgical procedure. A curved incision is given on the palate starting from just behind the maxillary tuberosity on one side and is carried medially along

the alveolar ridge up to the canine region. The same incision is carried out even on the opposite side. A “U” shaped palatal flap is elevated. This flap is elevated up to the edge of the hard palate.

The greater palatine neurovascular bundle is preserved at all costs. The soft palate is now retracted posteriorly and superiorly exposing the posterior edge of hard palate. This area is the area for dissection. The posterior edge of hard palate is taken down using kerrison’s punch or drill. The

nasal mucosa is exposed. This mucosal flap is lifted posteriorly till the choana is reached. The posterior portion of nasal septum and lateral superior nasal wall is also taken down. A stent is placed. The mucosal flap is then redraped in position.

Complications of transeptal approach:

1. Pressure necrosis of columella

2. Plugging of stent

3. Displacement of stent

4. Palatal dehiscence

5. Maxillary hypoplasia causing malocclusion

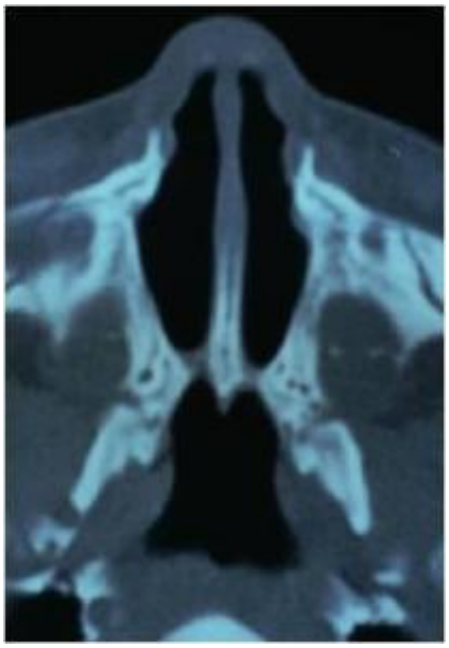
6. Granulation tissue formation around the stents.

Conclusion:

The main aim of choanal atresia repair is maintenance of nasal airway at all costs.

Success of choanal atresia surgery is determined by the necessity of post op dilatation of the chonal orifice or revision surgery on the same patient due to dense re stenosis.

Endoscopic view of choanal atresia



CT scan showing bilateral choanal atresia

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October 2012

Dentigerous cyst From supernumerary teeth

Dentigerous cyst

From supernumerary teeth

October 3, 2012 · Rhinology

Authors

Balasubramanian Thiagarajan

Abstract

Dentigerous cysts are the most common developmental odontogenic cysts. They are usually derived from the epithelial remnants of tooth forming organs. These cysts increase in size gradually. There may also be associated bone resorption. Managing these lesions creates problems in children. It is always better to be conservative in managing this problem in children because dentition is yet to complete in them.

Introduction:

Dentigerous cysts are the most common developmental odontogenic cysts. They are usually derived from the epithelial remnants of tooth forming organs[1]. Dentigerous cysts are classically defined as cystic lesions that are caused by separation of follicles from around the crown of unerupted teeth. Most commonly dentigerous cyst involves lower 3rd molar (mandibular) [2]. Dentigerous cysts were earlier termed as “Follicular cysts” since it was assumed that these cysts were derived from tooth follicle which is a mesodermal structure. Later this term was abandoned as it was conceived on an erroneous perception. Dentigerous cysts can also be caused by:

1. Impacted teeth

2. Supernumerary teeth – Is defined 8 as teeth in excess of usual configuration of 20 deciduous and 32 permanent teeth. Dentigerous cysts arising from supernumerary teeth accounts for nearly 5% of all these cysts.

3. Ectopic teeth (eruption of a teeth in sites other than the natural position). Most commonly seen ectopically erupted teeth involves 3rd molars

4. Rarely a tooth / root of teeth may be found in the sinus cavity. This teeth may have dentigerous cyst associated with it [3].

Theories of dentigerous cyst formation:

Usually all dentigerous cysts arise from the enamel organ after completion of amelogenesis. Dentigerous cyst arises due to accumulation of fluid causing separation of enamel of the unerupted tooth. The fluid present inside the cyst is hyperosmolar due to the presence of albumin, immunoglobulin and squamous epithelial debris. This hyperosmolar fluid causes influx of extracellular fluid into the cyst causing huge expansion of cyst to occur. The epithelial lining of the cyst secretes collagenase and osteoclast activating factor which causes local bone resorption causing further increase in the size of the cyst. This enlarging cyst encloses the crown of the unerupted teeth and is attached to its cemento-enamel junction.

Theories explaining genesis of Dentigerous cyst:

1. Theory of stimulation

2. Theory of inflammation[4]

Incidence:

Studies reveal that dentigerous cyst constitute more than a quarter of all jaw cysts. It predominates during the 2nd – 3rd decades of life[5]. There is a very slight male preponderance.

Majority of dentigerous cysts involves the mandibular third molar while maxillary canine is the next in the order of involvement. Very rarely dentigerous cyst can occur from ectopically erupted tooth within the maxillary sinus[6].

Symptoms:

These patients usually present with painless slow growing swelling involving the affected area. This swelling is very frim on palpation indicating cortical expansion. If it is present in the upper jaw then the swelling could involve the hard palate also.

These cysts are usually painless and dormant. There may be some degree of expansion of cortical bone. Presence of pain and rapid swelling definitly indicates inflammation. Fistula can rarely occur when the dentigerous cyst is present in the maxillary sinus. These patients usually present with evidence of sinusitis[7]. When these cysts are aspirated then yellowish fluid 9 could be observed. The swelling may also reduce in size following aspiration only to increase in size later.

Histology:

Histopathological examination of the cyst wall showed the cyst to be lined by reduced enamel epithelium. Connective tissue stroma will show features of primitive type of ectomesenchyme. Findings would depend on whether there is inflammatory component to the cyst is present or not. In non infected cysts the lining epithelium is 2-4 layers thick formed by primitive ectomesenchyme. These lining cells are low cuboidal to columnar. Retepegs could be seen only in cysts which are infected. The connective tissue stroma is loose and is rich in acid mucopolysaccharides. When the dentigerous cyst isinflamedthen it is characterised by the presence of hyperplastic rete ridges and the cyst wall demonstrates inflammatory infiltrate.

Theories of dentigerous cyst formation:

Intrafollicular theory:

According to this theory cyst formation occurs due to fluid accumulation between the layers of inner and outer enamel epithelium after crown formation.

Enamel hypoplasia theory:

This theory suggests that dentigerous cyst formation occurs due to degeneration of stellate reticulum at a very early stage of tooth development. There is also associated enamel hypoplasia.

Main’s theory:

This theory suggests that impacted tooth exerts pressure on the follicle with resulting obstruction of venous outflow. This induces rapid transudation of fluid across the capillary walls. This causes an increase in the hydrostatic pressure exerted causing separation of crown from the follicle. This may be associated with reduced enamel epithelium.

Radiographic features:

In plain radiographs these cysts present as a well defined unilocular radiolucency. Often there is a

demarcating sclerotic border. Since the cyst lining is derived from reduced enamel epithelium this

radiolucency preferentially surrounds the crown of the teeth. A large dentigerous cyst may provide an impression as if it is multilocular. This appearance is due to the persistence of bony trabeculae within the radiolucency. These cysts are particulary unilocular in nature.

Radiographic types of dentigerous cysts:

1. Central variety: In this variety the radiolucency surrounds the crown of the unerupted teeth. The crown can clearly be seen projecting into the cyst lumen.

2. Lateral variety: In this variety the cyst develops laterally along the tooth root, partially encircling the crown

3. Circumferential variety: The cyst entirely surrounds the unerupted teeth. Radiologically the unerupted teeth could be seen within the cyst cavity.

Dentigerous cyst showing unerupted teeth

CT scan showing dentigerous cyst

Treatment:

The usual treatment of dentigerous cyst is careful enucleation of the cyst in toto. Unerupted tooth if present should usually be removed along with the cyst. Sometimes if eruption of this teeth is Sometimes orthodontic treatment may be advocated to assist eruption of unerupted teeth.

Dentigerous cyst

Marsupialization:

This is more conservative method than enucleation of the entire cyst. This should be considered as the first line of management in children with dentigerous cyst. Major advantage of this procedure is loss of viable permanent tooth buds can be prevented. These patients should be followed up carefully by performing radiological imaging every 6 months in order to keep an eye on potential recurrence. This follow up should be continued atleast for a period of 2 years following marsupialization.

Unerupted teeth seen inside dentigerous cyst

Why dentigerous cysts should be treated?

1. They block eruption of normal teeth

2. They increase in size and cause displacement of teeth

3. They can cause bone destruction

4. They can cause displacement of vital structures like inferior alveolar nerve

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March 2012

Deviated Nasal Septum and its management A straight nasal septum is rather rare

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A straight nasal septum is rather rare

March 21, 2012 · Rhinology

Authors

Balasubramanian Thiagarajan

Deviated Nasal Septum and its management

Introduction:

Nasal cavity is divided into two portions by the presence of amid linenasal septum. The nasal septum has two components i.e. Bony andcartilaginousones. Bony components of nasal septum include:

1. Perpendicular plate of ethmoid superiorly – It articulates with the cribriform plate of ethmoid. Traumatic manipulation of perpendicular plate of ethmoid can lead to CSF leak. If this portion of nasal septum is the cause for nasal obstruction then it should be removed by sharp dissection rather than by twisting and pulling it out.

2. Vomer – Inferoposterior portion of nasal septum is formed by this bone. It is a keel shaped bone extending from sphenoid bone posteriorly and superiorly from the nasal crests of maxilla and palatine bone.

3. Nasal crest of palatine bone in the posterior portion of nasal septum

4. Nasal crest of maxilla forming the inferior portion of nasal septum

5. Anterior nasal spine forms antero inferior most portion of nasal septum

6. Nasal spine of frontal bone forms the antero superior portion of nasal septum

Cartilaginousportion of nasal septum include:

1. Septal cartilage forming anterior portion of nasal septum

2. Medical crus of alar cartilage

3. Vomeronasal cartilage: This thin strip of cartilage lies between cartilaginous nasal septum and the vomer

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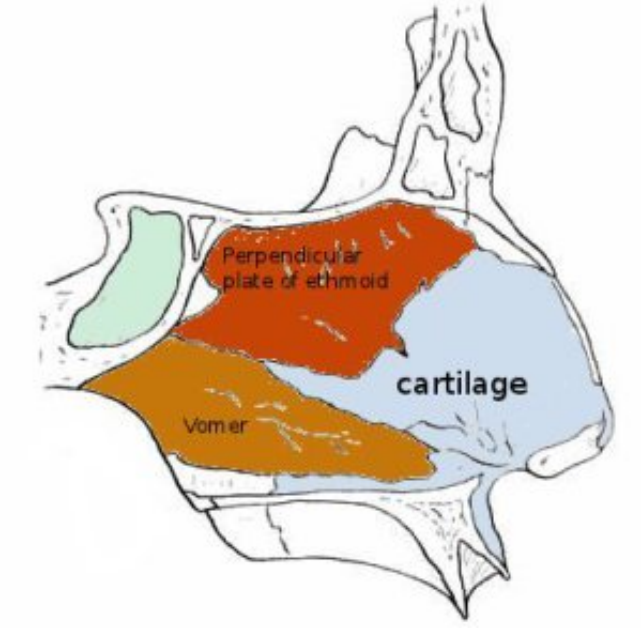
Fig. 1: Anatomy of nasal septum



Septal deviation:

Varying degrees of septal deviations have been documented at birth. These deviations have a tendency to accentuate as the child grows [1]. A dot central nasal septum is a clinical curiosity. Eventhough septal deviations are common they are usually not severe enough to cause symptoms [2].

Etiology of septal deviation:

Direct trauma – Many septal deviations are a result of direct trauma and this is frequently associated with damage to other parts of the nose such as fractures of nasal bone. Fractures involving nasal bones are the commonest fractures involving the facial skeleton. Nearly 40% of nasal septal fractures are unidentified during closed reduction of fractures involving nasal bones. These unrecognized / untreated septal fractures usually cause septal deviations at a later date [3]. Fractures involving nasal bones are rather uncommon in children under the age of 5. The incidence of nasal bone fractures progressively increases as the child grows older. The peak occurs around the age of 30 [4]. Studies stress the importance of identifying septal mucosa tear during clinical examination of these patients. Almost all patients with septal mucosal tear following nasal bone fracture invariably have associated fractures of nasal septum also.

Fig. 2: Nasal deformity with septal deviation following trauma

Birth moulding theory – Many patients with septal deviation do not give history of trauma. Birth moulding theory was propounded by Gray. According to him abonromal intrauterine posture may result in compression forces acting on the nose and upper jaws. Displacement of septum can occur in

these patients due to torsion forces that occur during parturition. Dislocations are more common in primipara and when the second stage of labour lasted for more than 15 minutes. Dislocations are generally to the right in the case of left occipitoanterior presentations and to the left with right occipitoanterior presentations. Subsequent growth of nose accentuates these asymmetries.

Differential growth between nasal septum and palate – This is the most acceptable theory today. When the nasal septum grows faster in certain individuals than the palate then the nasal septum starts to buckle under pressure.

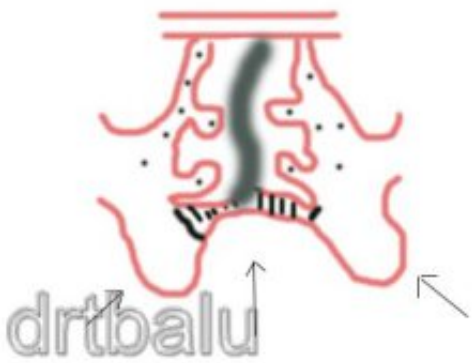


Fig. 3: Buckling of nasal septum

Figure showing unusual growth patterns between nasal septum and palate causing buckling of nasal septum

Pathophysiology:

Deformity of nasal septum may be classified into:

1. Spurs
2. Deviations
3. Dislocations

Spurs – These are sharp angulations seen in the nasal septum occuring at the junction of the vomer below, with the septal cartilage and / or ethmoid bone above. This type of deformity is the result of vertical compression forces. Fractures that occur through nasal septum during injury to the nose may also produce sharp angulations . These fractures heal by fibrosis that extend to the adjacent mucoperichondrium. This increases the difficulty of flap elevation in this area.

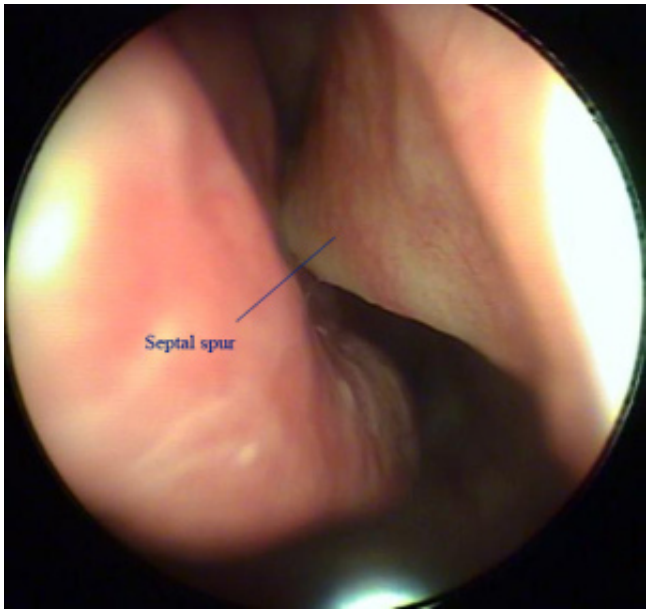


Fig. 4: Septal spur

Endoscopic view

Deviations – May be C shaped or S shaped. These can occur in either vertical or horizontal plane. It may also involve both cartilage and bone.

Dislocations – In this the lower border of the septal cartilageis displaced from its medial position and projects into one of the nostrils.

In patients with septal deviation a compensatory hypertrophy of the turbinates and bulla may occur on the side opposite to the deviation. If compression forces are involved the septal deviations are often asymmetrical and may also involve the maxilla, producing flattenting of the cheek, elevation of the floor of the affected nasal cavity, distortion of the palate and associated orthodontic abnormalities. The maxillary sinus is usually slightly smaller on the affected side.

Anterior septal deviations are often associated with deviations in the external nasal pyramid. Deviations may affect any of the three vertical components of the nose causing:

1.Cartilaginousdeviations

2. The C deviation

3. The S deviation.

Cartilaginousdeviations:

In these patients the upper bony septum and the bony pyramid are central, but there is a dislocation / deviation of thecartilaginousseptum and vault.

The C deviation:

Here there is displacement of the upper bony septum and the pyramid to one side and the whole of the cartilagenous septum and vault to the opposite side.

The S deviation:

Here the deviation of the middle third (the uppercartilaginousvault and associated septum) is opposite to that of the upper and lower thirds. With deviations of the nose, the dominant factor is the position of the nasal septum, hence the adage ‘as the septum goes, so goes the nose’. The first step, therefore in treating the twisted nose is to straighten the septum, and if this objective is not achieved, there is no hope of successfully straightening the external pyramid.

Effects of septal deviation:

Nasal obstruction – This is always found on the side of the deviation, and can also be present on the opposite side as a result of hypertrophic changes of the turbinates.

Mucosal changes – The inspiratory air currents are abnormally displaced and frequently gets concentrated on small areas of nasal mucosa, producing excessive drying effect. Crusting will occur and the separation of the crusts often produces ulceration and bleeding. Since the protective mucous layer is lost the resistance to infection is reduced. The mucosa around a septal deviation may become oedematous as a result of Bernouilli’s phenomenon. This oedema further increases nasal obstruction.

Fig. 5: Components of nasal septum

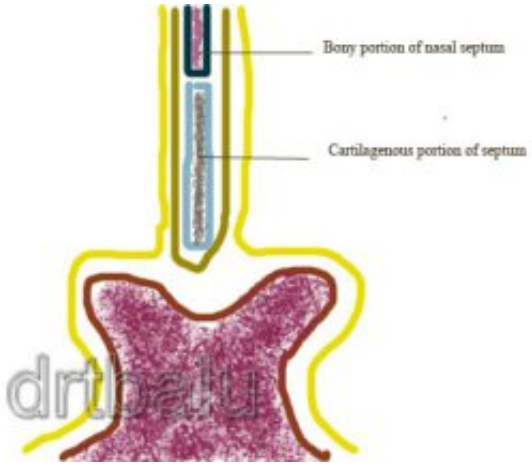


Fig. 6: S shaped deviation of nasal septum causing external deviation of nose

C shaped deviation of nasal septum causing external deviation of nose

Neurological changes – Pressure may be exerted by septal deviations on adjacent sensory nerves can produce pain. This was first explained by Sluder and the resultant condition became known as 'the anterior ethmoidal nerve syndrome'. In addition to these direct neurological effects, reflex changes perhaps may result from septal deformities which affect the nasopulmonary and nasal reflexes.

Symptoms :

The symptoms caused by septal deviations are entirely the result of their effects on nasal function. The dominent symptom being nasal obstruction, but this is rarely severe enough to cause anosmia.

Signs :

Septal deviations are evident on anterior rhinoscopy. This should be done without the use of nasal speculum because the insertion of speculum is sufficient to straighten the nasal septum. When the tip of the nose is lifted septal deviation become evident. Nasal obstruction may also be present on the opposite side (paradoxical nasal obstruction). This is due to the presence of hypertrophied turbinates. If the hypertrophy is limited to turbinate mucosa alone then it will shrink when decongestant drugs are used in the nasal cavity. If the hypertrophy is bony then deconstant drops is useless.

Septal deviations in the region of the nasal valve area cause the greatest obstruction, since this is the narrowest part of the nasal cavity. This can be identified by the cottle test. A positive cottle test will confirm the fact that narrowing is present in the nasal valve area. This is done by asking the patient to pull the cheek outwards and this manuver is supposed to open up the area thus reducing the block. The septum should not be considered in isolation and it is necessary to do a careful examination of the lateral wall of the nasal cavity. When ever sinus complications like sinusitis is suspected due to obstruction to the drainage channel of the sinuses by the deviation xray sinus must be taken.

Septal deviation in new born is associated with asymmetry of the nostrils, an oblique columella and tip which points in the direction which is opposite to the deviation. Most of these patients are diagnosed by the use of Gray's struts. These struts are 4mm wide and 2mm thick and after lubrication, are inserted into the nostrils and then gently pushed backwards along the floor of the nasal cavity, hugging the nasal septum. Normally these struts can be introduced for a distance of 4 – 5 cms, but in cases of septal deviation a frank obstruction is encountered, usually 1 – 2 cms from the nostril.

Role of Imaging:

CT scan can play a role in identifying septal fractures. These fractures may not be evident in routine radiographs of faciomaxillary regions.

Since fractures involving nasal bones have been implicated as common cause for septal deviations classification involving nasal bone fractures will have a bearing on management modality.

Stranc's classification of nasal fractures [5]:

Lateral oblique:

Unilateral nasal bone fracture with depression of bone

Unilateral depression and lateralization of the contralateralnasal bone

Bilateral nasal bone involvement with fracturing of thefrontal process of the maxilla

Frontal Type:

1: Does not extend posterior to a line drawn from thelower nasal bones to the

maxillary spineType

2: Flattening of the cartilaginous and bony structures,septal fractures,and intranasal mucosal injuries Type

3: Severe collapse of the nasal bones and upper lateralcartilages with telescoping of the septum. Associatedintracranial and orbital injuries may occur

Cottle has classified septal deviations into three types :

Simple deviations: Here there is mild deviation of nasal septum, there is no nasal obstruction. This is the commonest condition encountered. It needs no treatment.

Obstruction: There is more severe deviation of the nasal septum, which may touch the lateral wall of the nose, but on vasoconstriction the turbinates shrink away from the septum. Hence surgery is not indicated even in these cases.

Impaction: There is marked angulation of the septum with a spur which lies in contact with lateral nasal wall. The space is not increased even on vasoconstriction. Surgery is indicated in these patients.

Mladina's classification of septal deviation[6]:

Type I: Mild anterior deviation not compromising nasal function. This presents as a unilateral ridge along the nasal valve area. It does not involve the whole length of nasal septum and is not in contact with the lateral nasal valve the nasal air way is not compromised in anyway.

Type II: Anterior vertical deviation compromising nasal airway. There is unilateral vertical ridge in the nasal valve area compromising nasal airway

Type III: Posterior vertical deviation. In this condition the unilateral vertical ridge lies next to the head of the middle turbinate

Type IV: S shaped septal deviation

Type V: Horizontal spur is present in the nasal septum always in contact with the lateral nasal wall.

Type VI: Type V deviation with a deep horizontal gutter in the opposite side

Type VII: Crumpled septum

Indications for submucous resection of nasal septum:

1. Marked septal deviation occurring behind the vertical line passing between the nasal processes of the frontal and maxillary bones. This deviation must be the cause for the patient's symptoms.

2. Closure of septal perforations

3. Source of grafting material

4. To obtain surgical access in hypophysectomy, and vidian neurectomy

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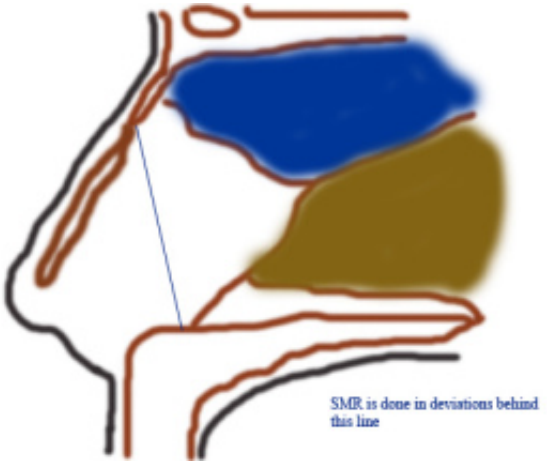
Figure showing in which type of deviation SMR should be done Surgically the septum is divided into anterior and posterior segments by a vertical line passing between the nasal processes of frontal and maxillay bones



Procedure:

Submucosal resection of nasal septum is ideally performed under local anaesthesia. 4% xylocaine is used as topical anesthetic agent by nasal packing. 2% xylocaine is used as infiltrative anesthetic agent. It is mixed with 1 in 1 lakh adrenaline. Infiltration is done at the mucocutaneous junction on both sides just behind the columella. The floor of the nasal cavity is also infiltrated on the concave side. Killian's incision is preferred for SMR operations. Killian's incision is the commonly used incision. It is an oblique incision given about 5mm above the caudal border of the septal cartilage.

The cartilagenous and bony nasal septum is exposed by elevation of mucoperichondrial and mucoperiosteal flaps on both sides. This is done by slicing the septal cartilage just above the columella to access the opposite side. Flaps are elevated on both sides of the nasal septum. the cartialge is fully exposed from both sides and is remove using a Luc's forceps or a Ballanger's swiwel knife. The flaps are allowed to fall back in place and wound is closed with catgut. Bony deviations along the floor of the nose if any are also chissled out before wound closure.

SMR should not be performed in children because it may affect growth.

Complications of SMR:

1. Septal hematoma
2. Septal abscess
3. Septal perforation
4. Nasal deformities due to excessive removal of dorsal strut of the septum
5. Removal of the columella cartilage will cause pig snout deformity

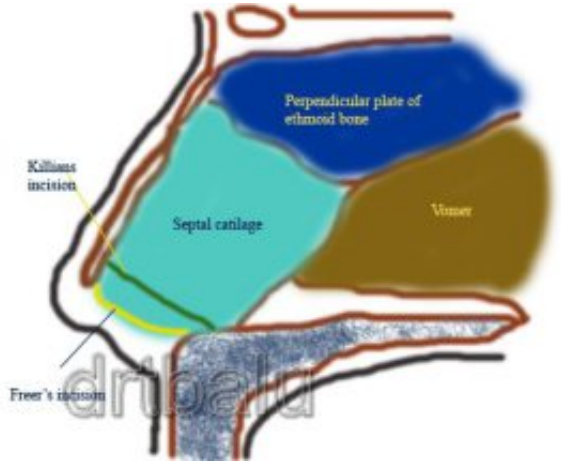


Diagram showing various types of incisions used in septal surgery

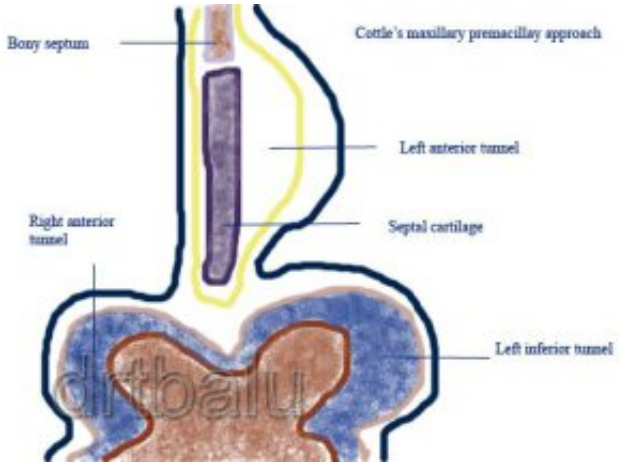


Figure showing various tunnels raised in a septoplasty surgery

Septoplasty:

This is a more conservative procedure. The anesthesia is the same as described for SMR operation. The incision is always sited on the concave side of the septum. Freer's hemitransfixation incision is preferred. This is made at the lower border of the septal cartilage. A unilateral Freer's incision is sufficient for septoplasty. Three tunnels are created as shown in the figure.

Exposure: The cartilagenous and bony septum are exposed by a complete elevation of a mucosal flap on one side only. Since flap is retained on the opposite side the vascularity of the septum is not compromised.

Mobilisation and straightening: The septal cartilage is freed from all its attachments apart from the mucosal flap on the convex side. Most of the deviations are maintained by extrinsic factors such as caudal dislocation of cartilage from the vomerine groove. Mobilisation alone will correct this problem. When deviations are due to intrinsic causes like the presence of healed fracture line then it must be excised along with a strip of cartilage. Bony deviations are treated either by fracture and repositioning or by resection of the fragment itself.

Fixation:

The septum is maintained in its new position by sutures and splints.

Advantages of Freer's incision:

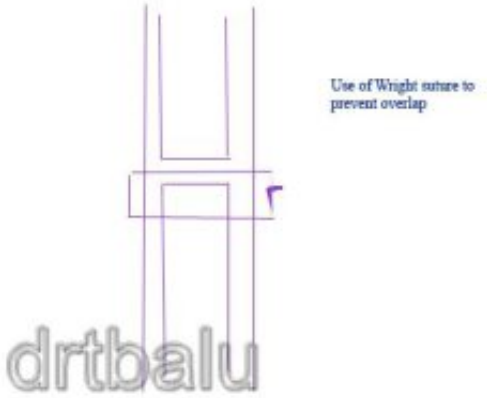
1. The incision is cited over thick skin making elevation of flap easy.

2. There is minimal risk of tearing the flap

3. The whole of the nasal septum is exposed.

4. If need arises Rhinoplasty can be done by extending the same incision to a full transfixation one.

Use of Wright's suture to prevent overlap



Advantages of Septoplasty:

1. More conservative procedure

2. Performed even in children

3. Less risk of septal perforation

4. Less risk of septal hematoma

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6. Clinical Manifestations in Different Types of Nasal Septal Deviation Yousif Ebrahim Chalabi etal The N Iraqi J Med December 2012;6(3) 24-29

September 2012

Evaluation of lacrimal apparatus

Evaluation of lacrimal apparatus

September 18, 2012 · Rhinology

Authors

Balasubramanian Thiagarajan

Abstract

Currently otolaryngologists have started doing dacryocystorhinostomy using nasal endoscopes. This procedure done by an otolaryngologist has its own obvious advantages. The first and foremost being the need for external incision has been dispensed with. On the flip side otolaryngologist is not conversant with the examination techniques involving this area. Even the standard text books of otolaryngology are woefully inadequate in details regarding this subject. This e book discusses the examination techniques and investigations pertaining to rhinolacrimal system

Lacrimal apparatus – Its evaluation

Introduction:

Dacryocystorhinostomy as a treatment modality for epiphora is commonly being performed endonasally using a nasal endoscope by otolaryngologists. It is hence imperative that they diligently examine the entire lacrimal system before proceeding with the procedure. Despite the commonality of this surgical procedure, standard otolaryngology text books contain fewer literature on this subject. History of surgery for nasolacrimal pathway obstruction dates back to Hamurabi 2200 B.C [1]. Dacryocystorhinostomy is the undisputed treatment of choice for lacrimal system drainage obstruction below the level of common canaliculi. Endonasal approach of dacryocystorhinostomy was first described by Caldwell in 1893 [2].

Epiphora can be caused by blockage of lacrimal drainage system / excess lacrimation / loss of lacrimal pump mechanism. Lacrimal pump mechanism could be disrupted due to lower lid laxity or weakness of orbicularis oculi muscle. Normal lacrimation / or excess of it can be caused by irritation to cornea / conjunctiva [3]. This reflex is initiated by stimulation of trigeminal nerve.

Trigeminal stimualtion can be caused by:

1. Corneal foreign body
2. Keratitis
3. Conjunctivitis
4. Ocular surface disorders (dry eye)

Epiphora:

Greek terminology meaning “Downpour”.

This is defined as excessive watering of eye. This is invariably caused by obstruction to tear drainage. Causes of epiphora include:

Congenital: Congenital nasolacrimal duct obstruction. Incidence varies between 1-6% [4]. It is beleived that massage of entire naso lacrimal system releives obstruction in more than 90% [5]of cases. Majority of these obstruction resolve during the first year of life hence urgent surgical management is not necessary [6]. Probing is also known to be beneficial in these patients. The time of probing is controversial. Probing is advised up to the age of 5 in these patients [7].

Acquired:

1. Primary acquired nasolacrimal duct obstructions
2. Dacryocystolithiasis
3. Orbital / lacrimal trauma
4. Canalicular lacerations
5. Actinomyces within the canaliculi – Actinomyces are anaerobic gram positive bacilli resembling fungi. These organism are normal commensal of oropharynx. These organism are capable of causing cast – forming canaliculitis 9 leading onto lacrimal tract obstruction.
6. Canalicular infections following herpes infections / ectropion – Viral infections constitute a well recognised common cause of acquired canalicular obstruction [8]. These patients give history of an episode of blepharokeratoconjunctivitis before epiphora. Antivirals (idoxuridine) which are prescribed for this condition too add to the woes by causing more lacrimal obstruction. Naso lacrimal obstruction caused by antivirals are transient and disappear after the drug is discontinued. Herpes simplex viral infections are known to cause punctal changes.

Clinical examination goal in these patients is to distinguish between epiphora and lacrimation. While epiphora needs to be surgically managed medically. The focus should be in differentiating anatomical obstruction from functional disorders.

Anatomical obstruction:

Obstruction to the lacrimal drainage system is the feature to look for in this condition. Pathological changes could be seen involving the lacrimal sac, irregularities in lacrimal drainage system (canalicular stenosis, canalicular blockage, obstruction to nasolacrimal duct, diverticulous formation etc.) Lacrimal pathways can be obstructed due to internal derangements like inflammation of the epithelial linining. This is known as intrinsic obstruction. If lacrimal pathways are affected by

deforming lesions from outside like tumors causing compression to it has been termed as extrinsic obstruction.

Physiologic dysfuntion causing epiphora:

This is also known as functional epiphora. Here there are no anatomical changes to the lacrimal pathway. The functioning lacrimal pump mechanism is at fault. Pump mechanism can be affected in conditions like eyelid malpositions, eversion of lacrimal punctum, poor orbicularis oculi muscle tone as seen in patients with Bell's palsy.

Grading of epiphora:

The commonly used grading system was devised by Sahlin [9].

Grade	Degree of Epiphora
0	No epiphora
1	Epiphora only outdoors and during windy times
2	Outdoor epiphora No indoor epiphora
3	Outdoor and indoor epiphora

Fig. 1: Grades of epiphora

Anatomy of lacrimal system an overview [10]:

The lacrimal system consists of a superior and inferior puncta at the medial ends of upper and lower eyelids. These two drain into upper and lower canaliculi. These two canaliculi join to form the common canaliculus. This zone is known as the upper lacrimal system. The common canaliculus inturn leads into the lacrimal sac. The sac is about 12 – 15 mm long. It eventually narrows and leads into the nasolacrimal duct which drains into the inferior meatus of the nose. The naso lacrimal duct is about 18 mm long. The sac and the duct comprise the lower lacrimal system.

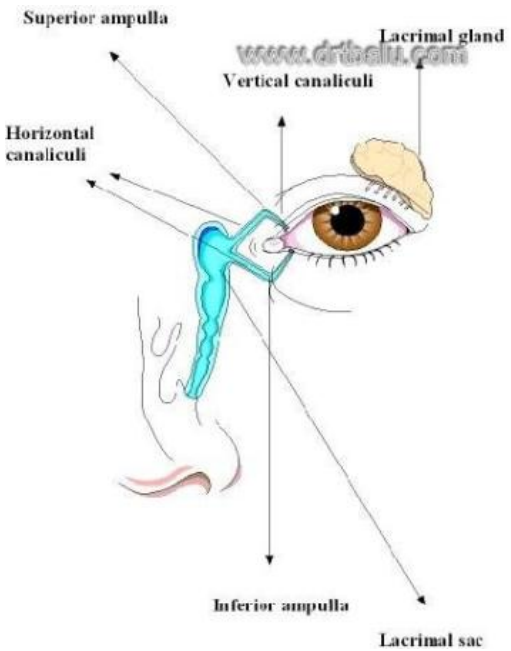


Fig. 2: Anatomy of lacrimal sytem

The junction between the common canaliculus and the lacrimal sac is guarded by the Rosenmuller valve. This valve prevents tear reflux. The nasal end of the nasolacrimal duct at the level of inferior

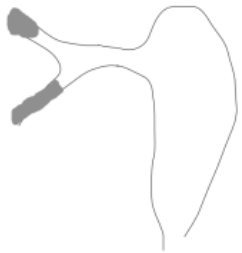
meatus is guarded by Hasner's valve.

Sites in the lacrimal system prone for obstruction:

Suprasaccal obstruction:

In this type obstruction lies proximal to the lacrimal sac. Obstruction can occur at the level of upper canaliculus, lower canaliculus and common canaliculus. Obstruction in these areas can occur following herpetic infections, taruma, irradiation.

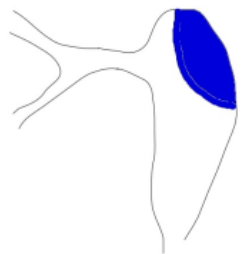
Suprasaccal obstruction



Saccal obstruction:

Here obstruction occurs at the level of lacrimal sac. This could be caused by tumors, diverticula, trauma etc.

Saccal obstruction

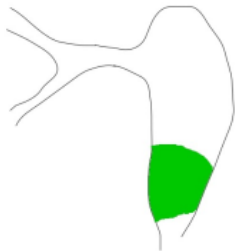


Subsaccal obstruction:

In this category the obstruction lies below the level of lacrimal sac. This condition commonly requires endoscopic dacryocystorhinostomy. This obstruction is more common than the rest.

Subsaccal incomplete obstruction

Complete subsaccal obstruction



Causes include:

1. Congenital nasolacrimal duct obstruction

2. Primary acquired nasolacrimal duct obstruction

3. Nasolacrimal duct obstruction following FESS

Functional obstruction:

This controversial term is used to explain those patients whose lacrimal system is patent to syringing but they still complain of epiphora. It should be borne in mind that the term obstruction

should be used only to indicate anatomical obstruction only.

Diagnosis of epiphora the Phylosophy behind it:

Theoretically speaking excessive tearing may be caused by -

Hypersecretion

Epiphora

Combination of both

Diagnostic evaluation should include:

1.Quantification of tear production

2.Assessment of nasolacrimal system patency

3.Differentiating epiphora from lacrimation

4.Defining the pathological process of epiphora

5.Differentiating anatomical from functional obstruction

6.Attempting to locate the obstruction in order to define the optimal surgical approach

Classification of Tests for lacrimal drainage pathway:

Anatomical tests

Functional tests

Secretory tests

Anatomical tests:

These tests are performed to locate the probable area of lacrimal tract obstruction. These tests include:

Palpation of lacrimal sac

Syringing / irrigation

Diagnostic probing

Dacryocystography

Nasal examination

CT / MRI

Functional tests:

These are performed to access the function of lacrimal apparatus under physiologic conditions.

This test is performed if there is no obstruction as evdenced by negative anatomical tests.

These tests include:

Flourescein dye disappearance test

Scintigraphy

Jones dye test I

Sacharin test

Tests of secretion:

These tests are performed to access secretory function of the lacrimal apparatus. These tests are performed in examining dry eyes. These tests include:

Schrimers test

Bengal Rose test

Tear-film break up

Tear lysozyme

Knowledge of various causes of lacrimation and epiphora really helps in clinical examination of these patients.

Excess lacrimation:

Supranuclear causes – Psychogenic / emotions

Stimulation of V cranial nerve – (Reflex tearing)

Lid causes (Blepharitis / Trichiaris)

Conjunctival diseases

Corneal diseases

Neuralgia

Ocular inflammation

Infranuclear causes – facial palsy, abberant innervation, crocodile tears

Lacrimal gland stimulation

Others – Bright lights, sneezing

Epiphora:

Functional insufficiency

Incorrect lid closure

Lid malposition

Punctal eversion

Punctal medialization

Anatomical obstruction

Combined lacrimation / epiphora – A combination of the above two categories

Facial nerve palsy – Corneal irritation and pump defects

Lower lid ectropion – Conjunctival irritation , ineffective pump mechanism

Thyroid diseases – Corneal irritation, defective canalicular function

Clinical history:

This is a very important aspect of lacrimal apparatus examination. This will provide vital clues to

the presence of canalicular disorders 12. History should include patient's present and past ophthalmological problems, nasal symptoms, medical and interventional relevant procedures also.

Unilateral tearing usually indicate obstructive pathology whereas bilateral tearing could be physiological. A child with a history of tearing since birth should arouse suspicion of membranous obstruction to nasolacrimal duct. Nasal disorders like nasal polyposis / sinusitis can also cause

unilateral epiphora.

Inspection and palpation should involve the following areas:

1. Eyelids

2. Medial canthus

3. Palpation of lacrimal sac

Eye lid examination:

Look out for lower eyelid laxity

Ectropion

Punctal eversion

Trichiasis

Blepharitis

Snap-back test – This test is performed by pulling the lower eye lid down and away from the globe and held for several seconds. On release the lower lid resumes its normal position. The time taken for resumption of normal position is noted. The patient should not blink during the test. This test provides an assessment of laxity of lower lid. The longer it takes for the lower lid to spring back to

position the more lax it is. This test is graded on a scale of 4 starting from 0. 0= normal and 4= lax lower lid.

Medial canthal laxity

Lateral canthal laxity

Orbicularis oculi muscle tone check

Pinch test – This test helps to assess orbicularis oculi muscle tension.

Examination of medial canthus:

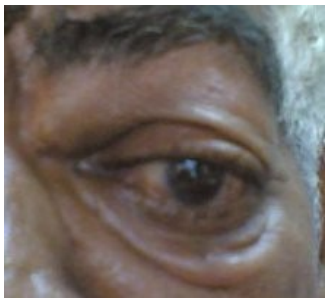
Lacrimal sac enlargement will be seen as mass below medial canthal tendon.

Enlargement above medial canthal tendon indicates neoplasm.

Palpation of lacrimal sac:

Normal sac is not palpable. Sac swelling is usually confined to below the medial canthal tendon. If there is neoplasm then it is likely to extend above the medial canthus.

Reflux of tears / pent up mucopurulent secretions can be seen on palpating the lacrimal sac area.



Swelling above medial canthus

Pain and tenderness over this area indicates acute dacryocystitis.

Sac being squeezed off its secretions

Sac swelling

Dye excretion tests:

These tests help in ascertaining drainage functions and patency of the entire nasolacrimal system.

Fluorescein dye is used for this purpose. This test is considered to be more physiological [11]since the lacrimal system is not instrumented and the dye flows along with tears through the normal passages. The principle of this test is evaluation of residual fluroescein dye in the eye following instillation of one drop of it into the unanaesthetized conjunctiva.

Caution: This test doesnot distinguish anatomical from functioning defects [12].

In performing this test one drop of 1% fluorescein is instilled into the lower fornix of each conjunctival sac. After 5 mins, the thickness of fluorescein of the tear meniscus is measured using cobalt blue filter. Studies reveal that it takes 5 mins for tears to normally drain through the system.

This test can safely be performed in infants and children [13].

Presence of fluorescein gives no information on the localisation of obstruction. Presence of residual

fluorescein is an indication for probing and syringing. When performing this test in infants the child should be held in a vertical position.

Dye test grading:

0=No fluorescein in the conjunctival sac

1=Thin flurescing marginal tear drop persists

2=More fluorescein persists somewhere between 1 and 3 grades

3=Wide brightly fluorescein tear strip

Among these grades 0 and 1 are considered normal

False negatives can occur in:

1. Large lacrimal sac

2. Mucocele

3. Distal nasolacrimal duct block

Break up time test:

This test is performed by placing a drop of fluorescein in the external canthus in the lower eyelid.

Its transport can be observed from lateral to medial across the eyelid and into each punctum. Holes in the tear film can be observed. This is break up time test.

Jones dye tests: This group of tests are used to distinguish between functional and anatomic outflow problems. The primary test is performed by placing topical anesthetic and flurescein dye into the conjunctival sac. Topical 4% xylocaine and oxymetazoline nasal sprays may be used to anesthetize and vasoconstrict the inferior meatus of the nose. A cotton tipped applicator is placed beneath the

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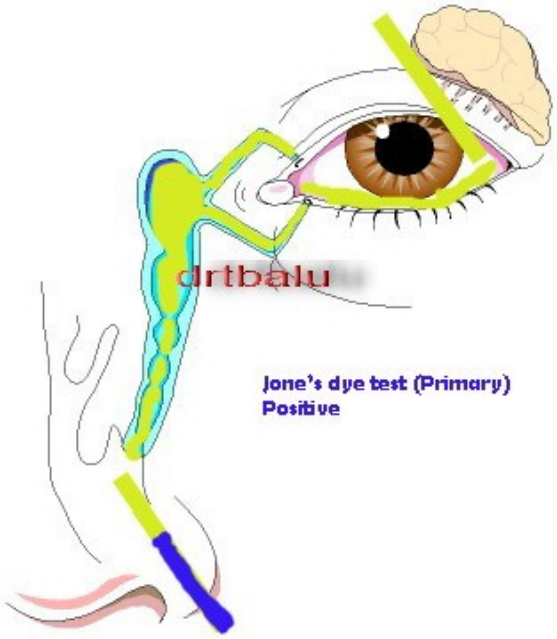
Jones dye test



inferior turbinate near the opening of the nasolacrimal duct. Recovery of fluorescein dye in the nose indicates a functionally and antomically patent system. Non recovery of the dye (negative result) suggests a functional or anatomic blockage.

In the event of negative dye test, secondary dye test should be performed. This test is performed after removal of residual fluorescein from the conjunctival sac. Clear saline solution is placed into the inferior canaliculus using a syringe / cannula. The irrigant is retreived from the nasal cavity by tilting the patient's head forward over a basin. If fluoresein dye is present in the irrigant (positive

result) then it is assumed that the upper lacrimal system is functional while the lower system is partially open and is not functional. Recovery of a clear irrigant (negative result) indicates a functional problem with the upper system.

Caution: This test is useless in patients with total lacrimal tract obstruction. This test should be performed only if the lacrimal system is patent for syringing.

Saccharin test:

This test is more or less similar to fluorescein dye test. This test is also hence physiological. A drop of saccharin is placed into anesthetized and the time taken for the patient to taste saccharin is measured. Approximate time is about 3.5 mins. The flip side in this test is that the patient should

have normal taste sensation.

Diagnostic probing and lacrimal syringing:

These are invasive tests. They provide valuable information on location of obstruction. They establish diagnosis of anatomical obstruction in the lacrimal system. This test is virtually useless in functional obstruction [14]. Syringing / irrigation of lacrimal system is not a physiological test since the pressures used is more than the normal pressure of lacrimal syste. Hence this test should be

interpreted with fluorescein dye test and clinical examination.

Procedure:

1. Topical 4% xylocaine drops applied to the conjunctiva

2. Punctum dilator is used to dilate the punctum and ampulla

3. A blunt cannula is placed in the inferior canaliculus. The lower eyelid is pulled down to straighten the inferior canaliculus. Superior canaliculus is gently stretched laterally prior to irrigation

4. Tip of the irrigator is placed in the inferior canaliculus, first vertically and then horizontally

with the eyelid on stretch. The tip is advanced 3-7 mm into the canaliculus and sterile saline

is injected.

5. It is important to avoid forced irrigation to avoid damage to the canaliculi

Interpretation: Regurgitation of irrigated saline through the opposite punctum indicates an obstruction in the common canaliculus or more distal structures. Regurgitation of fluid via the same canaliculus indicates punctal obstruction and syringing should be repeated via the opposite canaliculus. Irrigation into the nose indicates normal drainage function. It does not rule out functional obstruction.

Probing (Diagnostic):

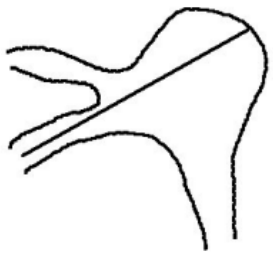
This test should be performed if syringing test indicate obstruction and the location of the obstruction is to be ascertained. Obstruction can be located in the canaliculi and their assessment is vital in deciding the mangement modality in these patients. If irrigated fluid regurgitates through

opposite punctum obstruction of common canaliculus or more distally is possible. The exact site in this scenario could be ascertained by careful probing of the entire system. Probing can be performed using blunt Bowman's probe which come in various sizes.

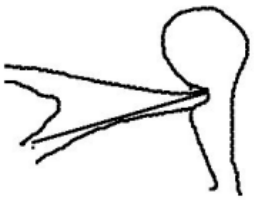
Procedure:

After instilling topical anesthetic drops into the conjunctiva the punctum is dilated using lacrimal

Fig. 3: Hard stop



Soft stop



probe. The probe is then passed vertically and then horizontally with the eyelid in stretch till the lacrimal bone is encountered or soft obstruction is reached. If the probe encounters lacrimal bone then it is known as hard stop. This is actually normal. If the probe encounters obstruction then it is known as soft stop. If irrigation showed reflux through the opposite punctum and the probe encounters hard stop then obstruction could be at the level of lacrimal sac or nasolacrimal duct.

Radiological evaluation:

Include:

Dacryocystography

Nuclear lacrimal scintigraphy

CT

MRI

Dacryocystography:

This is an anatomical investigation. This is indicated when there is block in the lacrimal system as indicated by syringing test. It helps in creating an internal image of the entire lacrimal system. In this test radio opaque water soluble dye is injected either into upper / lower canaliculus and magnified images are taken. Using digital subtraction techniques excellent images of the entire lacrimal system can be ensured.

Radiologic criteria of lacrimal pathology [15]:

1.Regurgitation of radio-opaque fluid into the conjunctival sac

2.Absence of fluid in the nose

3.Fluctuation of lumen of lacrimal system

4.Irregularity in contrast

5.Deformation involving lacrimal sac

Interpretation of irrigation and probing results

	Liquid in nose/pharynx	Reflux into lower canaliculus	Reflux into upper canaliculus	Result
Hard stop	yes	No	No	Normal
	No	No	Yes	Nasolacrimal duct stenosis
	No	No	No	Subsaccal obstruction
Soft stop	No	Yes	No	Medial canalicular/common canalicular block
	No	No	Yes	Distal common canalicular block

Nuclear lacrimal scintigraphy:

This is a non invasive physiological test. This test utilizes radiotracer technitium-99M pertechnitate. This can be analysed using a gama camera. This is useful only in patients whose lacrimal system is patent on syringing despite epiphora. This is found to be useful in difficult cases

and incomplete obstruction.

This test is performed without instilling topical anesthesia. A drop of technetium-99m is instilled into each conjunctival sac of a patient sitting in front of a gamma camera. Normal blinking of eyes are allowed. Patient stares at a distant target during a 20 mins test period while images are being

recorded with a gamma camera.

CT scan/ MRI scan:

Helpful in identifying tumors involving sac, or adjacent areas.

Rhinosporidiosis of lacrimal sac

Secretory tests:

These patients are useful in evaluating those with complaints of dry eye.

Schirmer's test: This test is basically prepared to quantitate tear production. This test is performed by placing strips of white filter paper 35×5 mm at the junction of the middle and lateral thirds of the lower eyelids after administration of a topical anesthetic agent. The tear production is measured

Schrimer's test

with the eyes closed. Produced tears will wet the filter paper. The length of the filter paper which becomes wet is assessed at the end of 5 minutes. Normal test result is between 10mm and 30 mm of wet filter paper. Normally it should not exceed 30 mm. A value of more than 30 mm is considered to be epiphora. A value of less than 10 mm is considered to be dry eye (hyposecretion).

Breakup time test:

This test indicates function of mucin layer / reflex hypersecretion of aquous component of the tears.

One drop of fluorescein is instilled into the external canthus of a lower lid and the patient is instructed to blink once and then to keep his eyes open. The holes developed in the tear film are observed at the cornea through a slit-lamp with illumination through the cobalt filter. The normal

breakup time should be approximately 15–30 s. A break-up time of less than 10 s indicates a deficiency and the epiphora should be treated with libricating eye drops .

Bengal Rose test:

This test is also similar to that of Break up time test. One drop of Bengal Rose dye is placed in the conjunctiva and the patient is instructed to blink several times within a minute. Interpalpebral

staining is seen in patients with dry eye.

Lysozyme lysis test:

The amount of a lysozyme activity and concentration is decreased in hypersecretion and in hyposecretion, and it usually precedes clinical symptoms. A lysozyme activity (and concentration)

is estimated on the basis of the inhibition of the growth of the bacterium Micrococcus lysodicticus.

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September 2012

Reducing bleeding during FESS

Reducing bleeding during FESS

September 10, 2012 · Rhinology

Authors

Balasubramanian Thiagarajan

Abstract

This article discusses the importance of anesthesia in endoscopic sinus surgery. Major aim of anesthetist in FESS should be to reduce blood pressure to such a level that bleeding is minimized. It also discusses the various steps that should be followed by surgeons to reduced intra operative bleeding during the procedure.

Introduction:

Bleeding is one complication that could increase the risk of complication during endoscopic sinus surgery.

Considerable amount of attention should be paid to reduce bleeding on the table during the surgical

procedure. Dry surgical field not only improves visibility during endoscopic sinus surgery, it also shortens the

duration of surgery. In this regard both anesthesiologist and the operating surgeon have a vital role to play.

Endoscope becomes rather useless when the operating field bleeds. Bleeding is more common if surgery is

performed on allergic / inflamed nasal mucosa. This is where operating surgeon should take extra precaution

in preparing the patient. Reduction of nasal allergy and inflammation is also known as mucosal preparation

prior to surgery. This is done by administering a course of antibiotic, antihistamine and topical steroid spray [1].

Ideally patient should be prescribed these medications at least 1 week prior to surgery.

Bleeding is more common close to large vessels. Stamberger[2] has included 4 areas which are responsible for

extensive bleeding during endoscopic sinus surgery.

1. Anterior ethmoidal artery located in an osseous channel close to ethmoid roof

2. Branch of sphenopalatine artery close to the posterior end of middle turbinate. This is more prone for

injury in patients with well pneumatized middle turbinate (concha bullosa)

3. Damage to sphenopalatine artery while attempting to widen the sphenoidal ostium

Classification of surgical bleeding during endoscopic sinus surgery:

Surgical bleeding during FESS has been classified into:

Arterial

Venous

Capillary

Out of these three types of bleeding it is the capillary bleed that causes the most trouble during FESS[3].

Capillary bleeding can be reduced considerably by careful packing of the nasal cavity with cotton pledgets /

neuropatties soaked in 4% xylocaine mixed with 1 in 10000 adrenaline. This concentration is being used by

the author with great degree of success. The concentration of adrenaline is the source of raging controversy.

One aspect should be clearly borne in mind, never exceed 7 ml of 4% xylocaine while packing. Any volume

about 7ml should prove to be toxic to the patient.

Position of the table:

This plays a vital role in reducing capillary bleed. If the surgical field is kept above the level of the heart blood

flow to the heart is considerably reduced. This is also known as postural ischemia. Systolic pressure has

been estimated to reduce by 2 mm of mercury for every 2.5 cms of head elevation[4]. Ideally during

endoscopic sinus surgery is the head of the patient is in an elevated position capillary bleeding will be reduced

a lot.

Maintaining normal body temperature[5]:

During surgical procedure maintaining normal body temperature is very important. Significant levels of hypo /

hyperthermia can affect platelet function causing increased bleed during the procedure.

Role of anesthesiologist:

Anesthesiologist play a vital role in reducing blood loss during surgery. Bleeding is directly proportional to the

mean arterial pressure. As long as the mean arterial pressure is held within a low range bleeding will be

minimal. Use of intravenous anesthetic agents like Propofol can reduce bleeding when compared to that of

conventional inhalational agents. Propofol is known to reduce brain metabolism and its circulation. Maximum

bleeding during Fess occurs from central vessels. Thus it plays a vital role in reducing bleeding. Use of

propofol with fentanyl supplementation actually serves the purpose. Even sevoflurane6 is known to increase

bleeding during Fess.

Use of Nitroglycerine infusion:

Nitroglycerine infusion during surgery causes prolonged hypotension. Only flip side to the use of NTG drip is

compensatory tachycardia which can push up the blood pressure. This tendency of compensatory

tachycardia is commonly seen in young individuals. This can be obviated by putting the patient on

preoperative night dose of beta blockers. Captopril can be used on the table to reduce the hear rate that

could occur due to NTG drip.

If the patient is under prolonged hypotension, their status should be meticulously monitored.

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Fracture frontal bone and its management

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Fracture frontal bone and its management

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Abstract:

Fractures involving frontal bone are rather uncommon. Injuries to this bone is rather critical because of its proximity to brain. This article attempts to discuss this topic with focus its prevalence, its causes and various treatment modalities available. Three crucial areas need to be addressed. They include anterior table, posterior table and frontal sinus outflow tract. Posterior table fractures are usually associated with anterior table fractures and CSF leak. Hence these patients should be treated as head injury cases

Introduction:

Fractures involving frontal bone is rather rare because of its protected location [1]. It is basically protected from trauma by the prominence formed by the nasal pyramid. Incidence of fractures involving this area ranges between 5-15% [3]. Fractures involving this bone is considered to be rather dangerous because of its proximity to brain as well as due to the cosmetic defects it can produce. The proximity of this bone to the orbit and naso frontal duct doesn't help matters either. Fractures involving this area if not treated promptly can lead to [2]:

1. Meningitis
2. Mucopyocele
3. Encephalitis
4. Cerebral abscess

It should be borne in mind that all cases of fractures involving frontal bone should be considered as a potential head injury and should be managed similarly because of its close proximity to the brain.

Causes of frontal sinus injuries:

1. Road traffic accident
2. Assault
3. Industrial accidents
4. Recreational accidents

Classification of frontal bone:

Anterior table fracture

1. With / without displacement
2. With / without outflow tract injury

Posterior table fracture commonly occurs in combination with anterior table fracture

1. With / without displacement
2. With / without dural injury / CSF leak
3. With / without outflow tract injury

Displacement is considered to be present if it is about the width of one table of the frontal bone.

Anatomy of frontal sinus:

Among the para nasal sinuses this sinus shows the maximum variations. In fact variations are so immense that it can safely be stated that frontal sinuses are unique in each and every individual. It may be absent in 5 % of individuals. It is more or less shaped like a L. Drainage channel of frontal sinus is highly variable.

Posterior wall: corresponds to the anterior wall of the anterior cranial fossa.

Floor: is formed by the upper part of the orbits. Frontal sinus appear very late in life. In fact they are not seen in skull films before the age of 6.

The sinus drains into the anterior part of the middle meatus through the fronto nasal duct.

Frontal outflow tract shows conglomeratization of air cells.

Types of frontal sinus air cells include:

I – Type I frontal cell (a single air cell above agger nasi)

II – Type II frontal cell (a series of air cells above agger nasi but below the orbital roof)

III – Type III frontal cell (this cell extends into the frontal sinus but is contiguous with agger nasi cell)

IV – Type IV frontal cell lies completely within the frontal sinus

Materials and methods:

In this study all patients with Faciomaxillary trauma presented at Stanley Medical College Hospital during the 4 year period from 2009 – 2012 were taken.

Inclusion criteria:

All patients with Faciomaxillary injuries.

Exclusion criteria:

Nil

Total number of patients enrolled: 128

All of them had Faciomaxillary injuries.

Number of patients with frontal bone fracture: 12

Total number of patients with Faciomaxillary trauma	Number of patients with frontal bone fracture
128	12

Anterior table fractures – 10

Posterior table fractures – 2

All 12 patients were males.

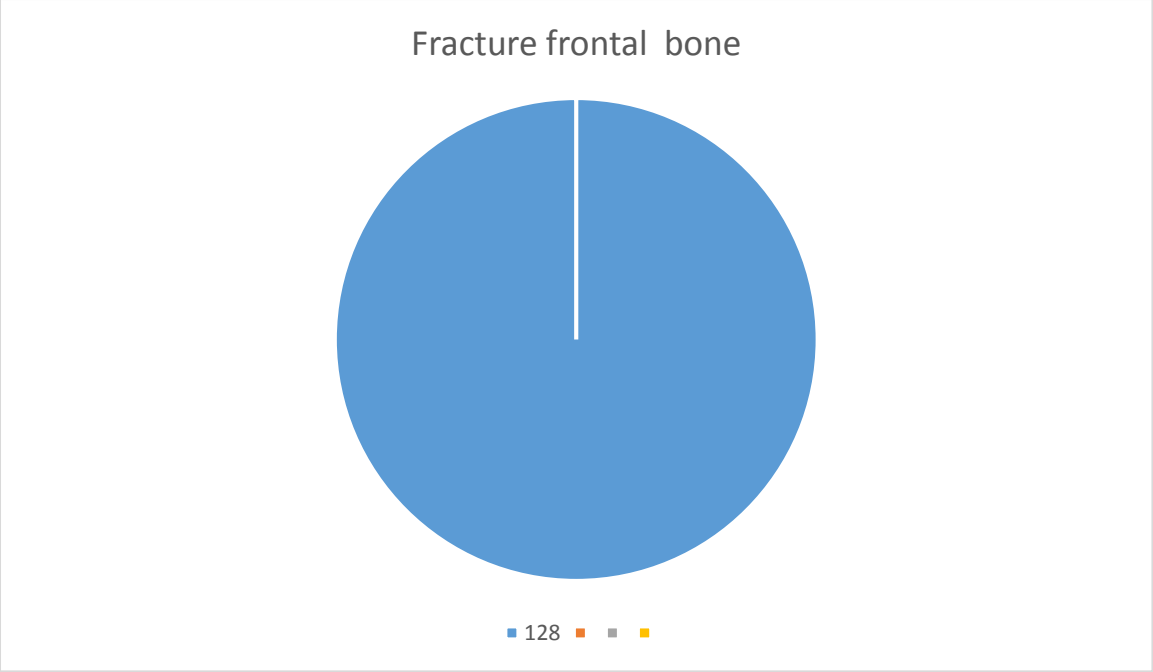


Chart illustrating patients with fracture frontal bone among those with facio maxillary trauma

Clinical photograph of a patient with fracture anterior table of frontal sinus

Common causes of fractures involving frontal bones:

1. Road traffic accident
2. Assault
3. Industrial accidents
4. Recreational accidents

A study of our patients reveal that 8 of them suffered injury due to road traffic accident and 4 patients suffered due to assault.

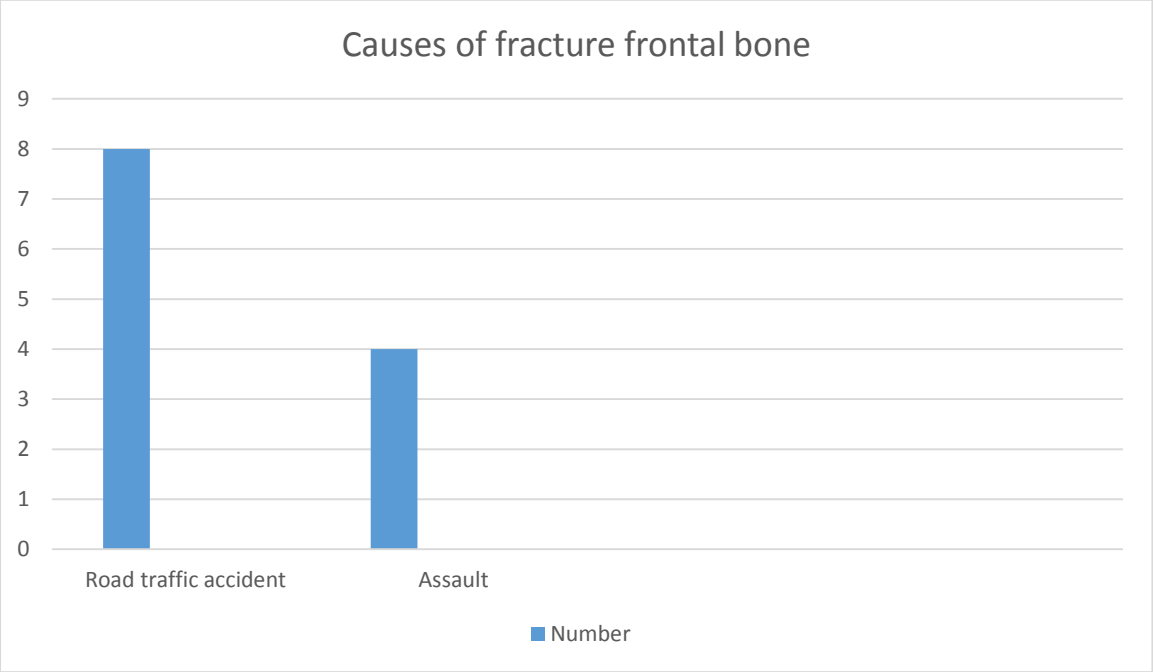


Chart depicting the common causes of fracture frontal bone in our study

Clinical presenting features:

These include:

1. Cosmetic defect
2. Headache
3. CSF leak (in patients with posterior table fractures)

Treatment goals:

1. Protection of intracranial structures
2. Control of CSF leak
3. Prevention of late complications like secondary mucoceles
4. Deformity correction

Assessment of patient with injury to frontal sinus:

1. All suspected patients should undergo a complete ophthalmic examination to rule out injury to the eye.
2. All these patients must undergo CT scan of brain and skull for compete evaluation
3. The patient's consciousness should be monitored carefully to rule out intracranial complications
4. Other associated injuries must be looked for because the force necessary to cause fracture of frontal bone is enormous.

Anterior table fractures:

This is caused by low energy trauma. Commonly this fracture is isolated non displaced fracture. If there is no displacement then observation alone is sufficient.

If it is associated with displacement of fractured fragments then open reduction with internal fixation is the way to proceed.

If there is associated damage to frontal outflow tract then frontal sinus obliteration is advised along with open reduction and internal fixation. Reconstruction of outflow tract is a difficult procedure and is unpredictable. One other way to tackle this problem is observation and medical management followed by endoscopic sinus surgery in future is need arises.

When open reduction with internal fixation is performed care should be taken to avoid entrapment of mucosa [4] within the bone fragments as this would lead to mucocele formation at a later date. Accurate identification of frontal out flow tract injuries are rather difficult to identify in routine CT imaging. Periodical CT scans can be performed to look for evidence of frontal sinus outflow tract obstruction. Frontal trephining can be performed

and endoscope can be introduced via the trephine to observe for evidence of outflow tract obstruction.

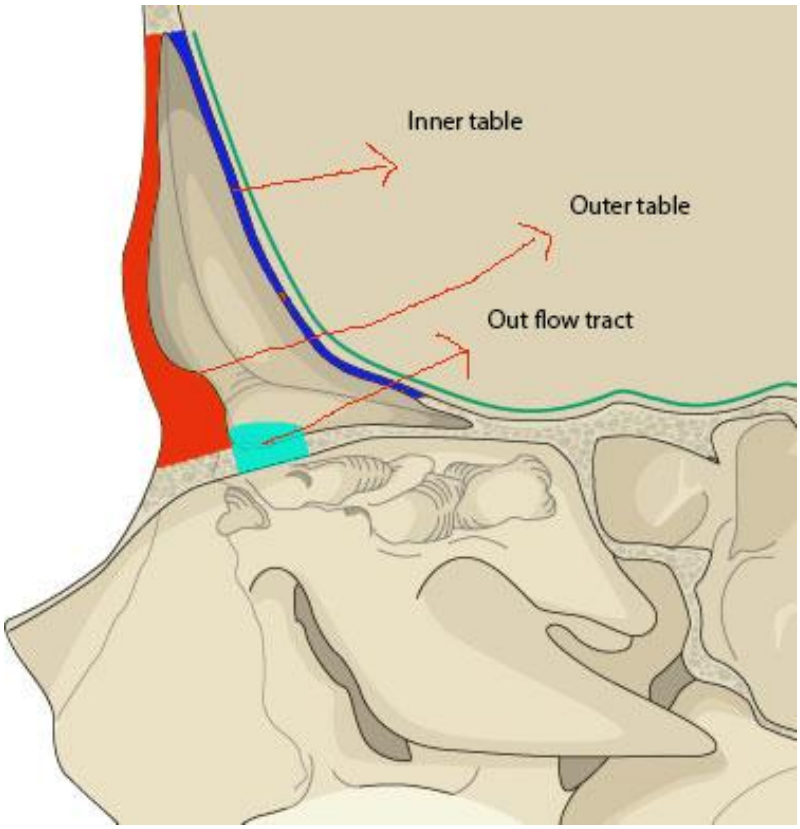
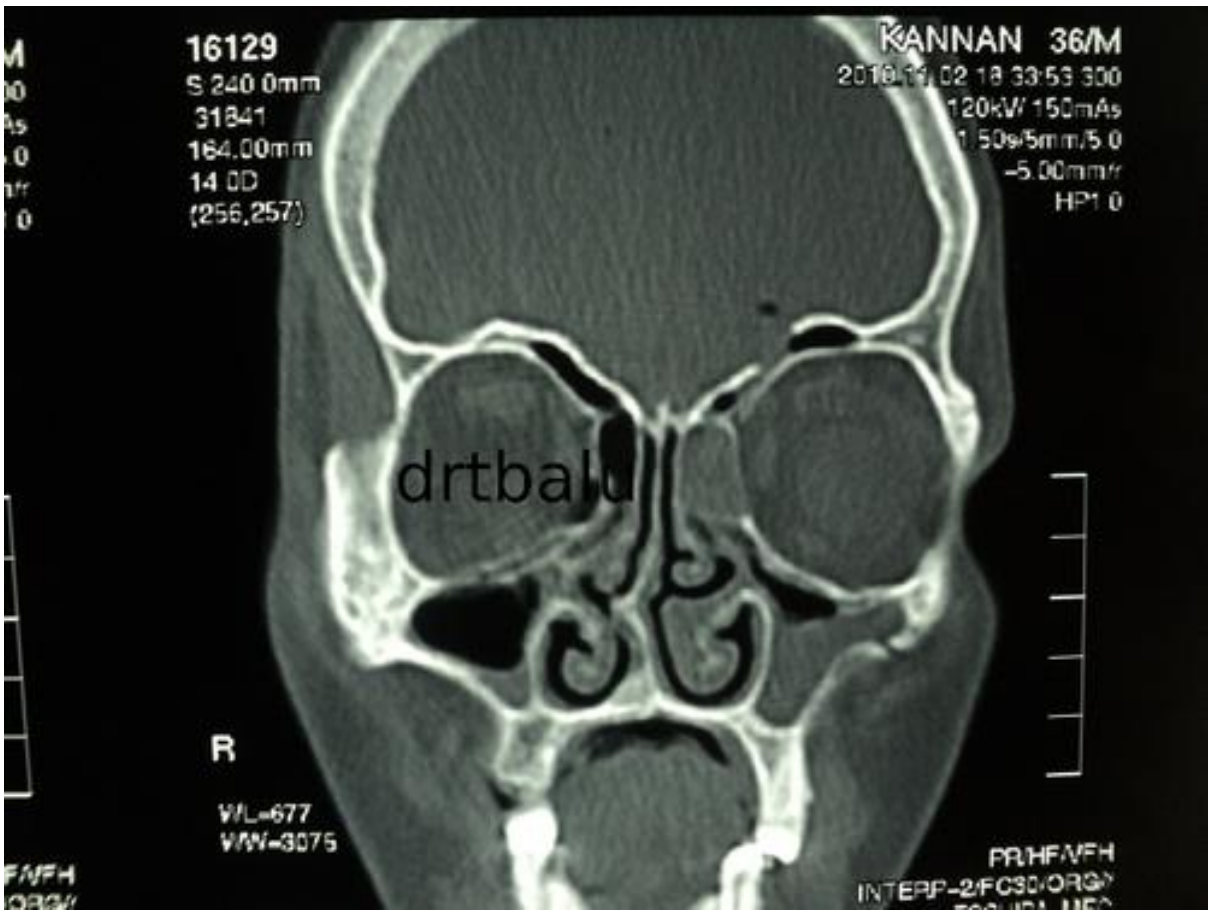


Diagram illustrating various parameters that should be focussed on during the management of frontal bone fractures

Three vital areas that should be addressed while managing fractures of frontal bone:

1. Anterior table

2. Posterior table
3. Frontal sinus outflow tract



Coronal CT scan of nose and sinuses showing fracture involving posterior table of frontal sinus

Management:

Our patients who needed open reduction and internal fixation were managed with

1. Supraciliary approach
2. Bicoronal approach [5]

Since one of the two patients who needed open reduction and internal fixation had a scar in the supraciliary area the incision was sited in the supraciliary region.

Image showing fractured fragment of outer table being exposed via supraciliary incision

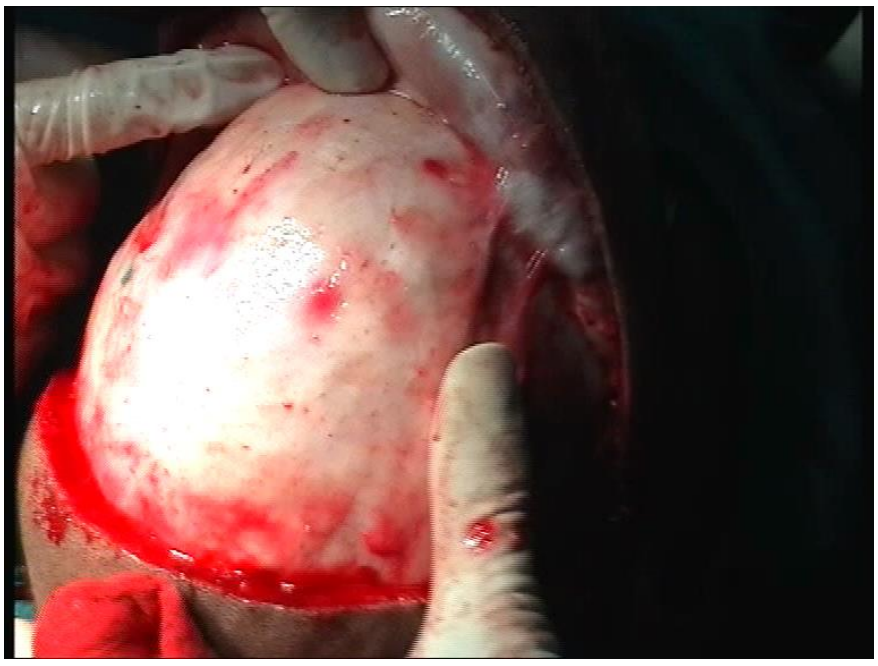


Image showing Bicoronal flap being raised

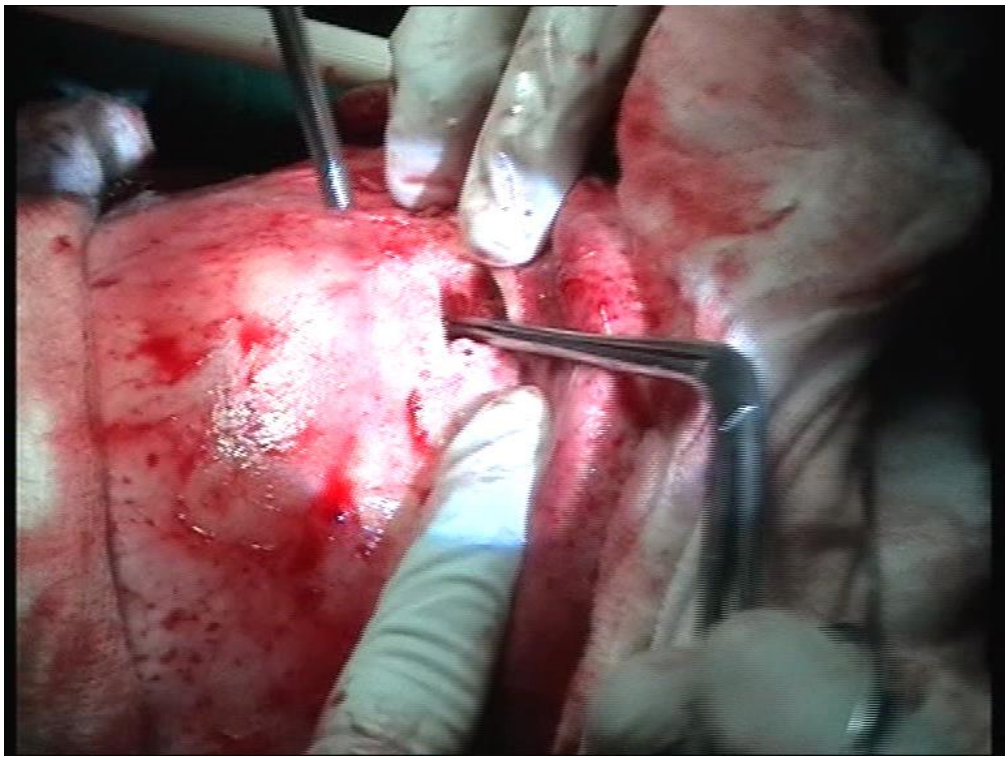


Image showing frontal sinus being entered via anterior table of frontal sinus

In our series only one patient presented with posterior table fracture with CSF leak. This patient was taken up for open reduction and CSF leak closure via Bicoronal approach. The lone patient who had fracture involving both anterior and posterior tables with frontal outflow tract block had a cannula placed in the frontal sinus under endoscopic guidance and left in place for 4 weeks.

Tips for frontal bone fracture repair:

1. All depressed fractures of more than one table width should be reduced
2. Titanium mini plates are very useful with good success rate
3. In comminuted fractures titanium mesh is ideal

Conclusion:

1. All frontal bone fractures need not be reduced
2. When there is posterior table fracture with CSF leak the patient should be treated as head injury patient
3. Depressed fractures with depression of more than the width of the frontal table alone need to be reduced
4. Frontal sinus outflow tract obstruction needs lot of expertise in management. It is always better to obliterate frontal sinus in these cases.

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Fracture zygoma and its management our experience

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Fracture zygoma and its management our experience

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Abstract:

Zygoma is a very crucial component which maintains facial contour. Fractures involving zygoma is very common, in fact it is the second most common facial bone to the fractured following facial trauma (next only to nasal bones). Fractures involving maxilla not only creates cosmetic deformities, it also causes disruption of ocular and mandibular functions too. This article attempts to discuss in detail the etiopathogenesis and the various management options available. It also includes our 3 years experience in treating these patients at Stanley Medical College Chennai. During the period of 3 years between 2010 - 2012 about 82 patients got treated in our institution for faciomaxillary trauma.

Introduction:

Zygoma plays a vital role in maintaining facial contour. This is because the facial contour is directly influenced by underlying bony architecture [1]. Fracture and dislocation of this bone not only causes cosmetic defects but also disrupts ocular and mandibular functions too. The zygomatic region is a prominent portion of the face next only to the dorsum of the nose. This predisposes this bone to various trauma [2]. The bony architecture of this bone is rather unique, it enables it to withstand blows with significant impact without being fractured. At the most it gets disarticulated along its suture lines. Fractures can involve any of the four articulations of zygoma which include zygomatico-maxillary complex, zygomatic complex proper, orbitozygomatic complex. Fractures involving zygoma should be repaired at the earliest because it can cause both functional and cosmetic defects.
Important functional defects involving this bone is restriction of mouth opening due to impingement on the coronoid process [3]. It is hence mandatory to diagnose and treat this condition properly. It is also important to reduce this fracture and fix it accurately, because skeletal healing after inadequate reduction can cause reduced projection of malar region of the face leading on to cosmetic deformities. Accurate assessment of position of the fractured bone should be performed in relation to skull base posteriorly and midface anteriorly. This assessment is very important before reduction is attempted to ensure accurate reduction of the fractured fragments.

Importance of facial buttresses in fracture of middle third of face [4]:

The buttress system of midface is formed by strong frontal, maxillary, zygomatic and sphenoid bones and their attachments to one another. The central midface contains many fragile bones that

could easily crumble when subjected to strong forces. These fragile bones are surrounded by thicker bones of the facial buttress system lending it some strength and stability.

Components of Buttress system:

For better understanding the components of the facial buttress system have been divided into:
1. Vertical buttresses
2. Horizontal buttresses

Vertical buttress:
These buttresses are very well developed.
They include:
1. Nasomaxillary
2. Zygomaticomaxillay
3. Pterygomaxillay
4. Vertical mandible

Majority of the forces absorbed by midface are masticatory in nature. Hence the vertical buttresses are well developed in humans.

Horizontal buttresses:

These buttresses interconnect and provide support for the vertical buttresses. They include:
1. Frontal bar
2. Infraorbital rim & nasal bones
3. Hard palate & maxillary alveolus

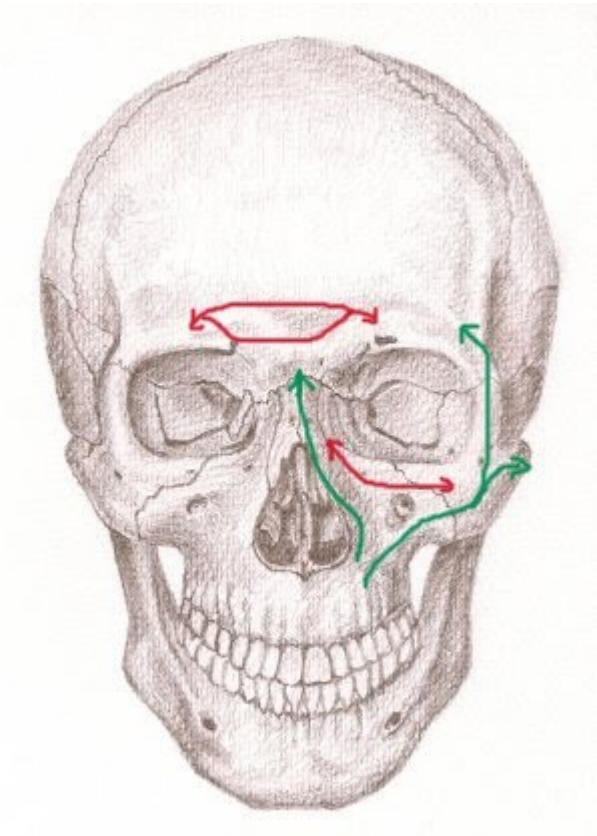


Diagram illustrating tbe Buttress system of the facial skeleton

Incidence:

Among the 280 trauma patients admitted for treatment at Stanley Medical College 82 had sustained faciomaxillary injuries.

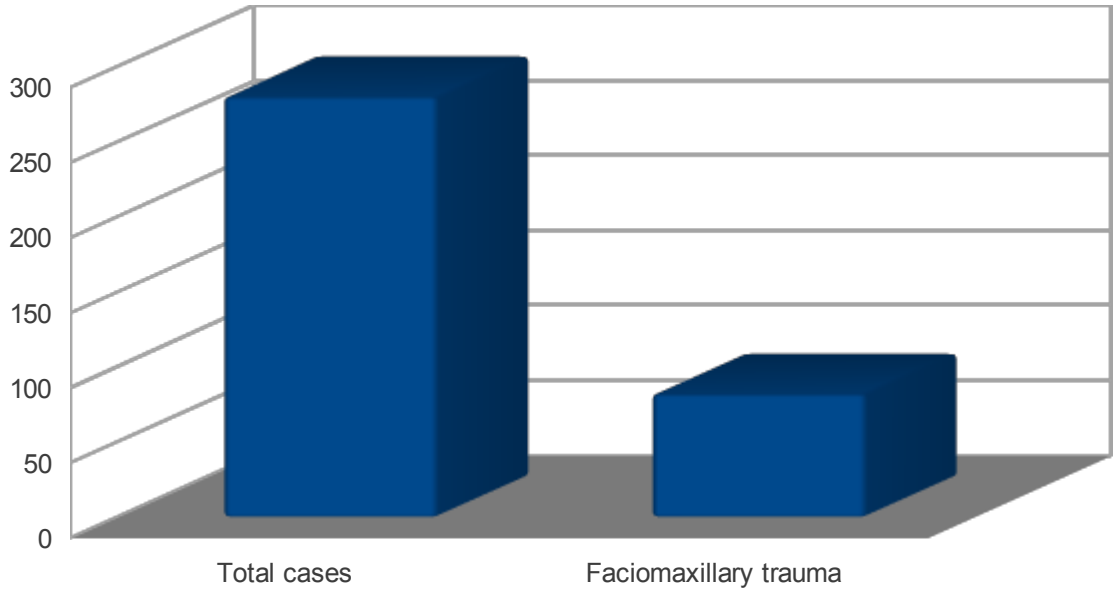


Chart showing the incidence of faciomaxillary trauma in comparison with other trauma cases

Among the cases studied 70 of them were males and the rest were females.

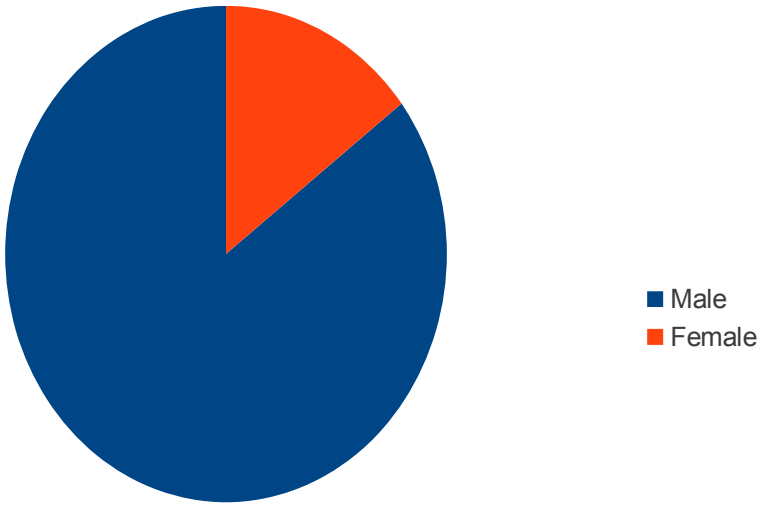


Chart showing the number of male and female patients who presented with fracture of zygoma

Classification of zygoma fracture:

Leefort classification:

1. Non displaced – Symptomatic treatment. No reduction necessary

2. Displaced – Closed reduction is necessary

3. Comminuted – Open reduction is necessary

4. Orbital wall fracture – If ocular symptoms predominate it should be attended first. After oedema subsides then open reduction can be attempted.

5. Zygomatic arch fracture – Open reduction with stablization using micro plates / wiring.

Knight & North classification [5]:

This classification suggested by Knight etal in 1961 helped to determine prognosis and optimal treatment modality for these individuals.

Group I fractures:

In these patients fracture lines in zygoma could be seen only in imaging. There is absolutely no displacement. These patients could ideally be managed conservatively by observation and by asking the patient to eat soft diet.

Group II fractures:

This group includes isolated fractures of the arch of zygoma. These patients present with trismus and cosmetic deformities.

Group III fractures:

This include unrotated fractures involving body of zygoma.

Group IV fractures:

This involves medially rotated fractures of body of zygoma.

Group V fractures:

This involves laterally rotated fractures of body of zygoma. This type of fracture is very unstable and cannot be managed by closed reduction. Open reduction will have to be resorted to.

Group VI fractures:

This is complex fracture. It has multiple fracture lines over the body of zygoma. This condition is difficult to manage by closed reduction. Open reduction and microplate fixation is indicated in these patients. This type of fracture should not be managed by closed reduction alone because the presence of oedema / haematoma would mask the cosmetic deformity giving an impression that reduction has occurred. After reduction of oedema and followed by the action of masseter the fractured fragment may distract making the cosmetic deformity well noticeable.

In our Institution patients with zygomatic bone fractures presented under various categories of Knight classification. Majority of them belonged to Group I and II fractures.

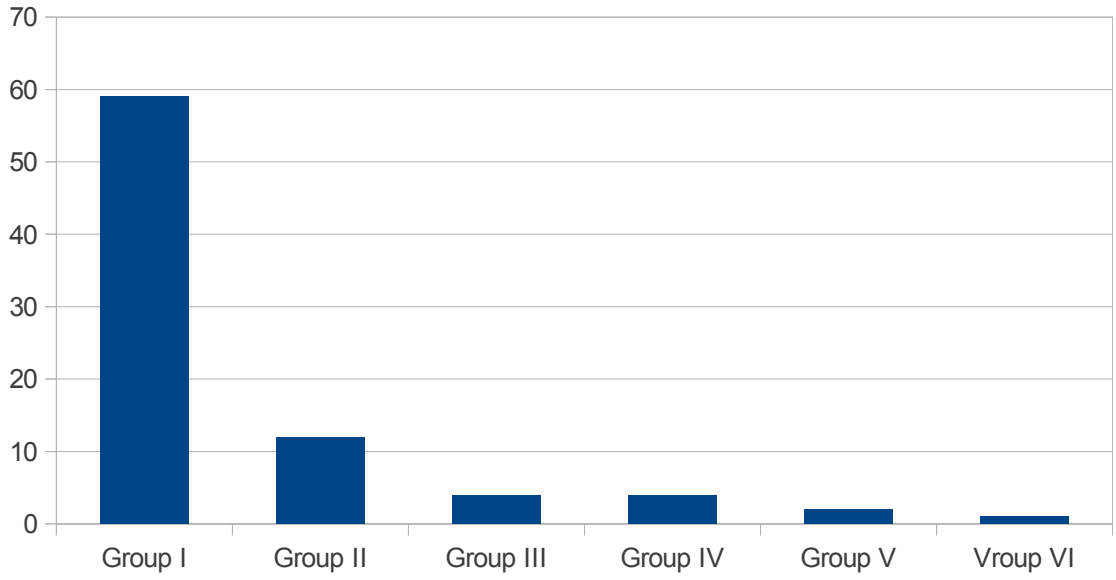


Table showing the number of patients in various groups of Knight's classification

Mason's classification of fracture zygoma:

Mason [6] etal used CT imaging to classify various forms of fracture zygoma. CT imaging provides the most accurate information about facial skeleton. Fractures involving facial bones, their positions, whether it is displaced or not can be clearly seen in CT scan images. Mason classified fractures involving zygoma into:

1. Low energy injury
2. Medium energy injury
3. High energy injury

Low energy injury:

Low energy fractures involving zygoma involves minimal or no displacement of fractured fragments. In this group of patients fractures are commonly seen in the frontozygomatic suture line. This area is very stable and hence fractures involving this area can be treated conservatively.

Middle energy injury:

Fracture zygoma due to middle energy injury causes fractures of all its supporting buttresses. There may be mild to moderate displacement and comminution. These patients invariably need eyelid / intraoral approach for adequate reduction and fixation of fracture.

High energy injury:

This injury frequently causes Lefort fractures. These patients have difficulty in opening their mouth. Repair of fractures involving this area should be carried out through multiple approaches which include:

Bicoronal approach

Intraoral approach

Eye lid approach

Studies reveal that primary bone healing allows quicker and stronger bone formation than callous healing [7]. Rigid fixation of fractured fragments promote primary healing in preference to callous formation.

While performing open reduction it should be borne in mind that Titanium plates [8] are preferred to biodegradable ones when the process of reduction leaves small gaps between fractured fragments.

Clinical features:

1. Anaesthesia / Paraesthesia of that side of the face

2. Inability to open the mouth

3. Flattening of zygomatic area

4. Diplopia

5. Subconjunctival haemorrhage

6. Eye lid oedema

7. Periorbital haemorrhage

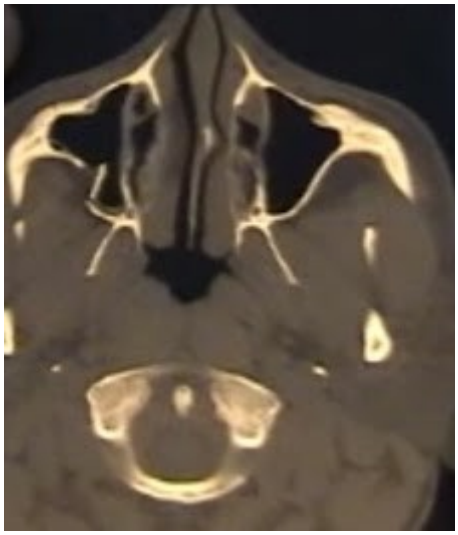
8. Lateral canthal dystopia

9. Ipsilateral epistaxis

10. Buccal sulcus haematomas

11. Enopthalmos in orbital floor fractures

Opthalmic examination is a must if any of the opthalmic manifestations of fracture of zygoma is seen. In the presence of ruptured globe, retinal detachment and traumatic optic nerve atrophy management of ophthalmic manifestations take precedence over fracture reduction procedure.



Axial CT image of nose and sinuses showing fracture of zygoma with medial displacement (stable)

Picture showing depressed fracture of zygoma (medial displacement)

Picture showing reduction being performed via intraoral route

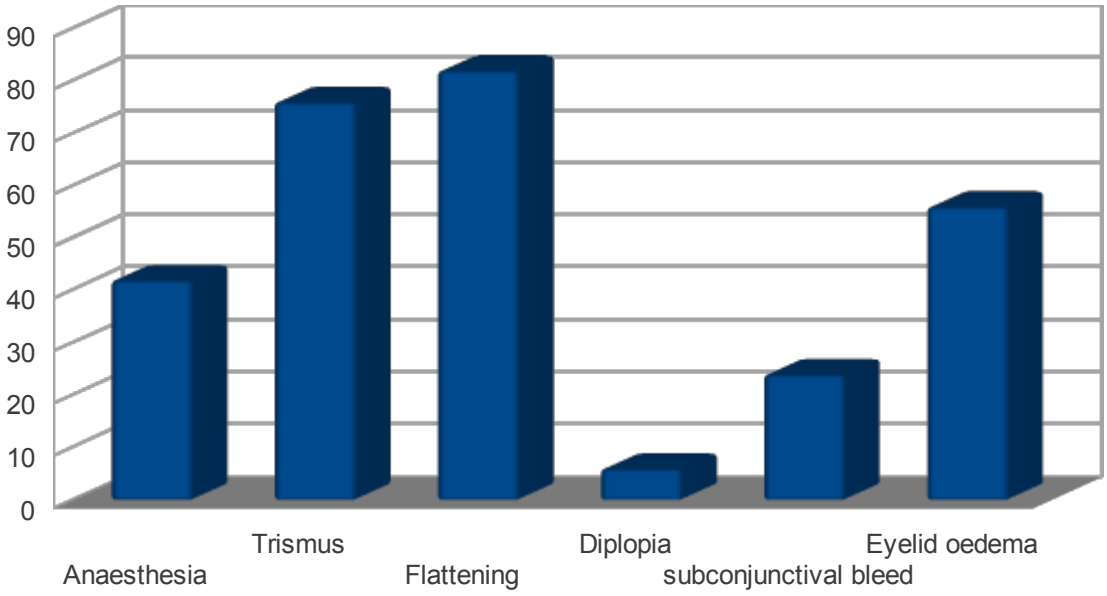


Chart showing the common clinical features our patients presented with

Orbital exploration is indicated in the following circumstances:

1. Severe comminution

2. Displacement of orbital rim

3. Displacement of greater than 50% of the orbital floor with prolapse of orbital contents into the maxillary sinus

4. Orbital floor fracture of greater than 2 cm^2

5. Combination of inferior and medial orbital wall fractures

6. Suspected involvement of orbital apex

Our patients commonly presented with cosmetic defect of the malar area, followed by trismus.

Isolated zygomatic arch fracture:

This fracture can be managed easily without the necessity of internal fixation / splinting if reduction is performed within the span of 72 hours following injury. Fractures involving zygomatic arch can cause inability of movement of mandible. These fractures can be reduced using Gillie's temporal approach or Dingman's[9] supraorbital approach. Other approaches include Buccal sulcus approach. Studies reveal that temporal or supraorbital approaches provided the best results [10].

Ruler test:

This is a rather useful clinical test to identify patients with fracture of zygoma.

Two rulers are used as shown in the figure below to perform this test. These rulers are placed in front of the ears. Ruler is found to deviate on the side of fracture.

Figure showing ruler test being performed

Gillie's technique of reducing fracture zygoma:

Small incision is made over temporal area superficial temporal artery is avoided.

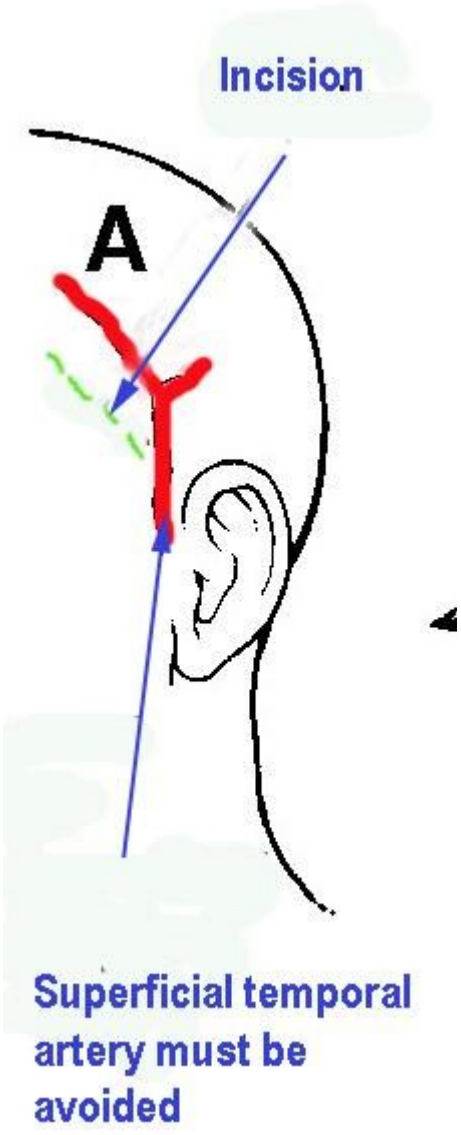
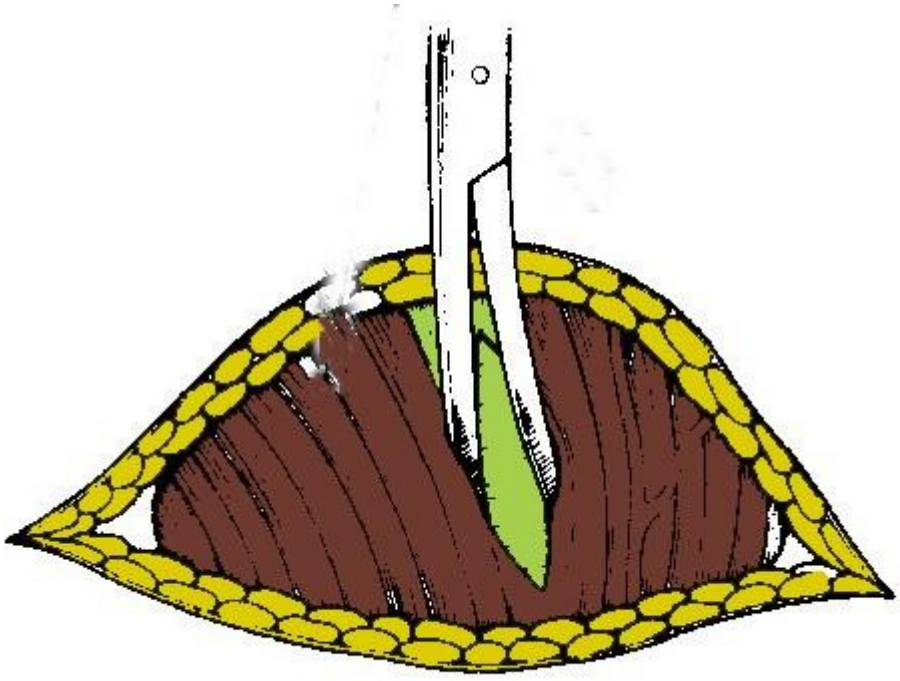
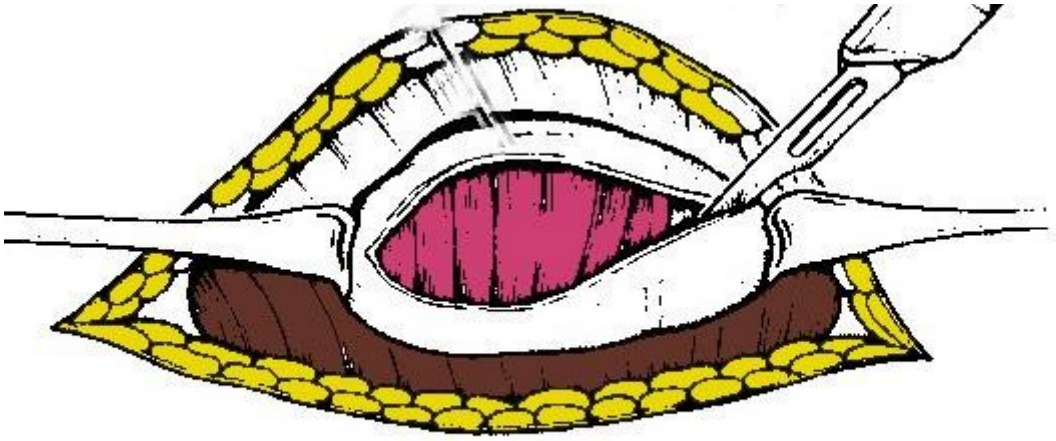


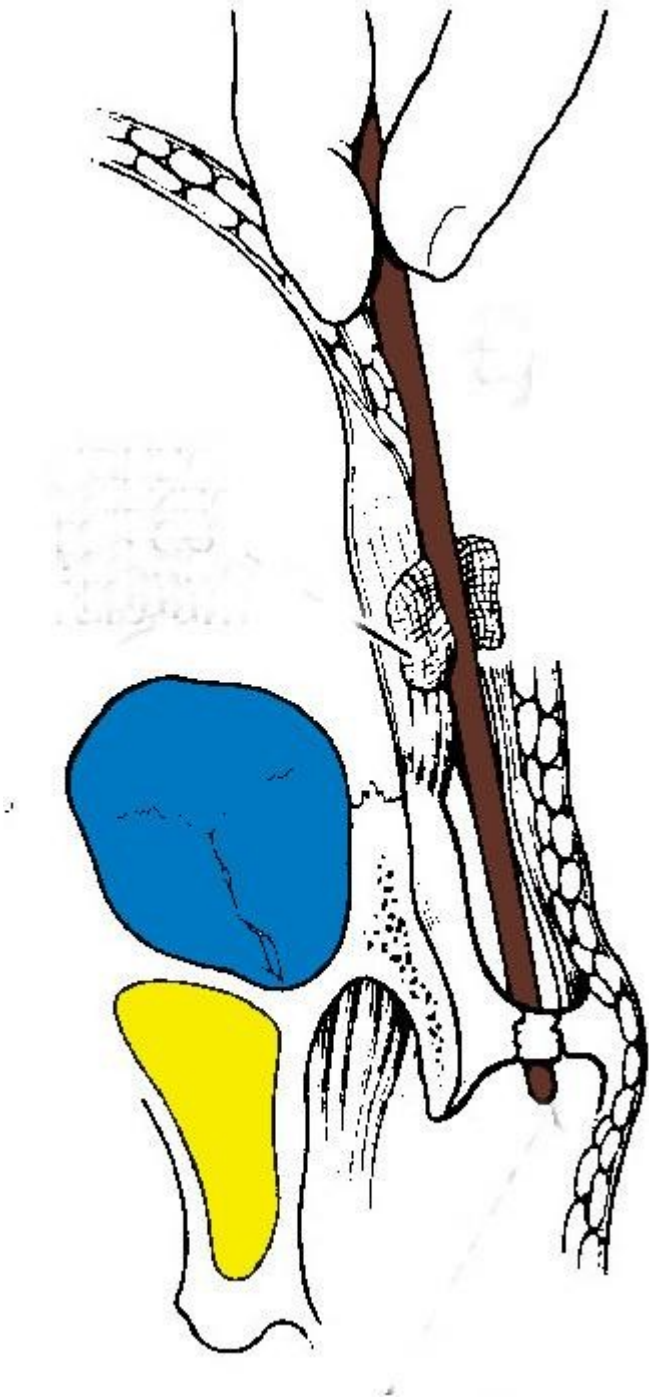
Figure showing incision for Gillies procedure



Auricularis superior muscle is cut along the line of its muscle fibers



Temporalis fascia is cut with a knife



Periosteal elevator is inserted through the incision and the fractured fragment is elevated. A gauze piece is used as a leverage

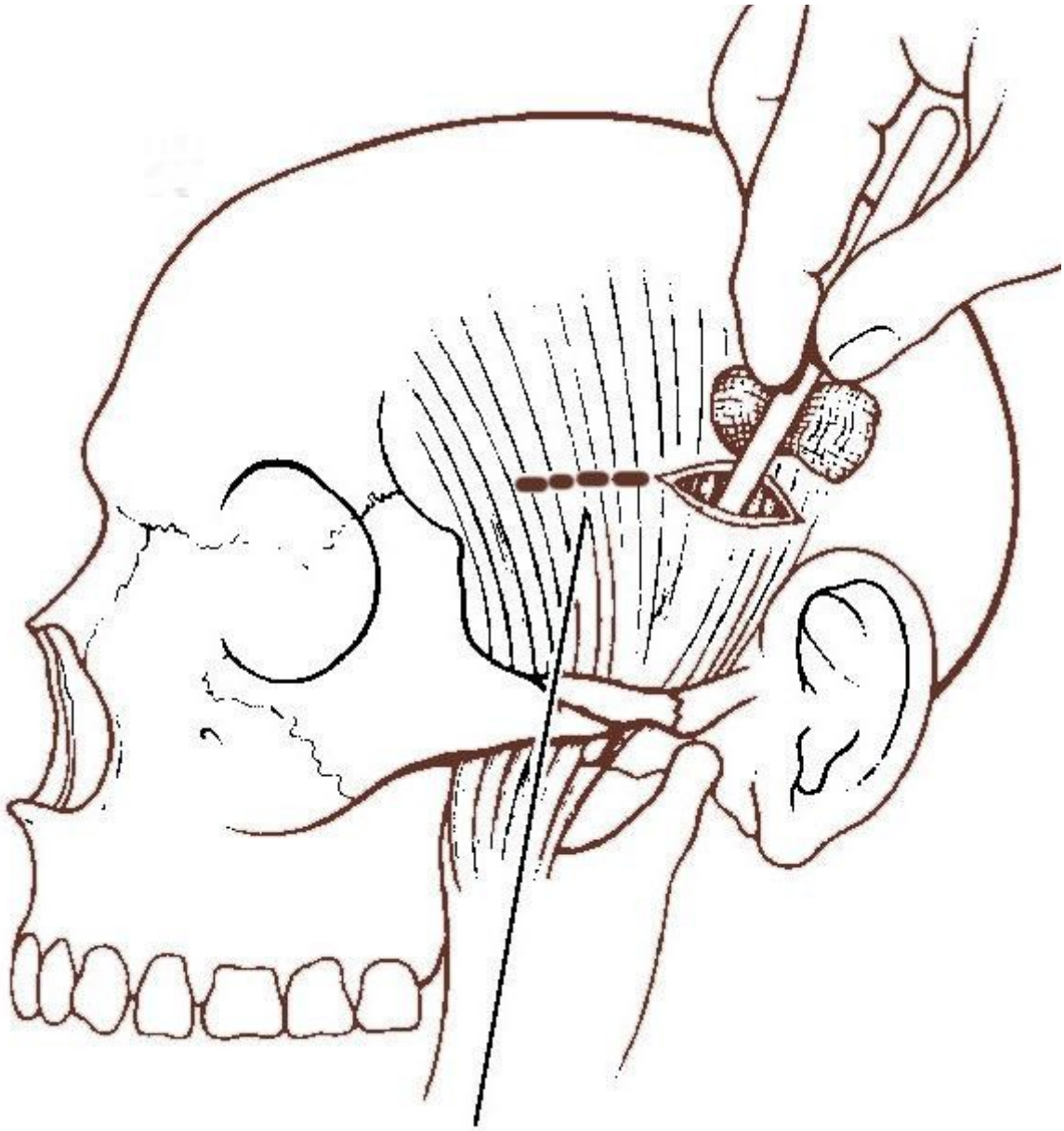


Figure showing fracture arch of zygoma being reduced.

Zygomatic complex fratures:

These fractures are invariably managed by open reduction with two point / three point fixation. Surgical procedure is performed usually after 4- 6 weeks following injury. If fractures are more than 3 months old then osteotomy will have to be performed. Bone grafts need to be used to perform accurate repair. Usually two point fixation is sufficient in majority of patients. Two point fixation involves microplate fixation at zygomatico-frontal and zygomatic arch areas. When using microplates for zygomatico-frontal area care should be taken to position it slightly posteriorly so

that untoward subcutaneous projection of the plate can be avoided.

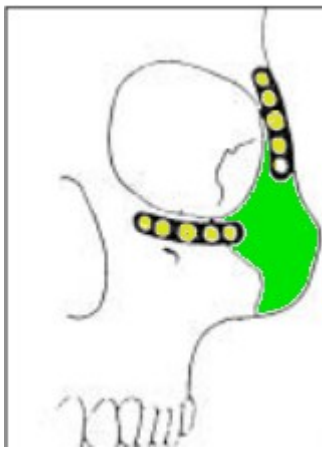


Figure showing two point fixation points

Two point fixation is sufficient in a majority of patients. Rarely when fracture is extensive and associated with lateral displacement of fractured fragments three point fixation need to be resorted to.

Bicoronal approach may be used to approach this area for open reduction purposes. Eye brow incision / transconjunctival incisions can also be used to access this area.

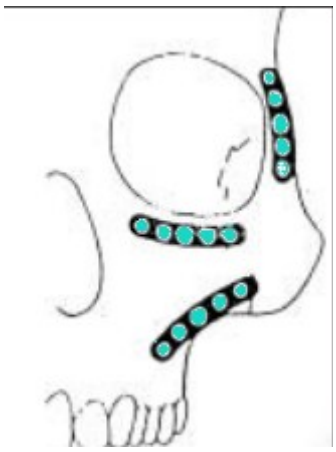


Figure showing three point fixation areas

As shown in the figure three point fixation includes fixing:

1. Frontozygomatic suture
2. Infraorbital rim
3. Zygomatico maxillary buttress

In our analysis only 2 of the 82 patients studied needed two point fixation. All the other patients were managed either conservatively or by closed reduction.

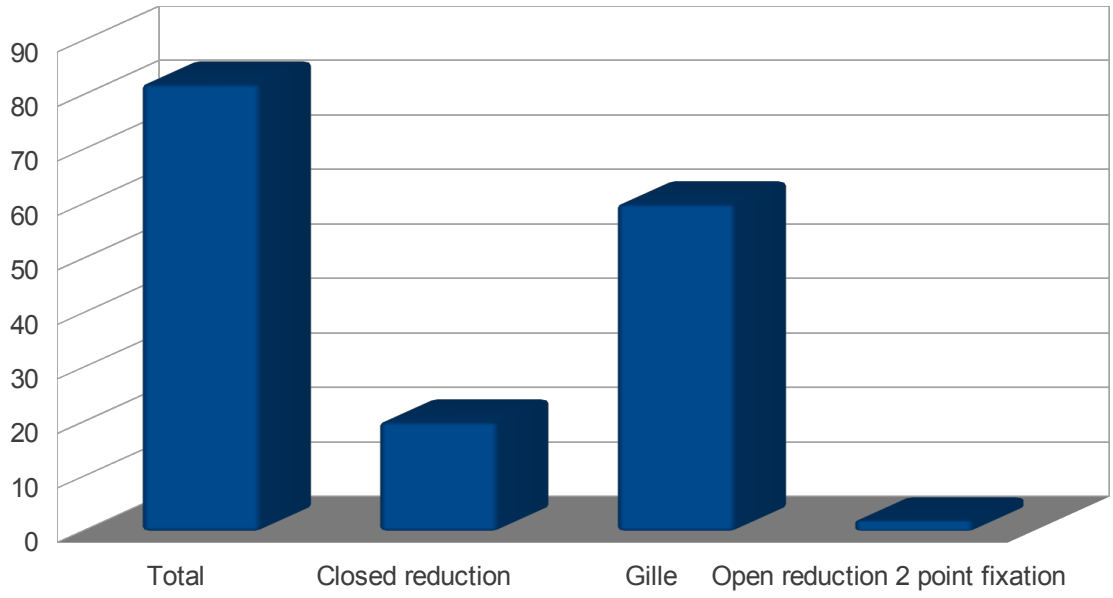


Chart showing the number of patients who under went various procedures for the management of zygomatic fracture

Classification of zygomatico-maxillary complex fractures:

Zingg's classification [11]:

Zingg in 1992 had separated zygomatico-maxillary complex into three types:

1. Type A

2. Type B

3. Type C

Type A :

This type is associated with one component of the tetrapod structure.

This type is subdivided into three subgroups:

Type A1 zygomatic arch alone is fractured.

Type A2 fracture of lateral orbital wall

Type A3 fracture of inferior orbital rim

Type B fracture:

This type of fracture involves all 3 buttresses. Also known as Tripod fracture. This fracture will have tobe treated by two point fixation / three point fixation techniques.

Type C fracture:

These are comminuted fractures involving zygoma.

Orbital floor is the weakest component of the zygomatic-maxillary complex. Type A3, B and C are associated with fracture of the floor of orbit with risk of injury to orbital contents.

Conclusion:

This study reveals:

1. Majority of our patients with fracture zygoma presented with flattened malar region. Next common symptom of presentation was trismus.

2. All of our patients except for one with fracture zygoma had stable medial displacement

3. Majority of our patients were managed conservatively / Gillie's procedure.

4. Only two patients needed open reduction with three point fixation

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July 2012

INFECTED HALLER CELL. RADIOLOGY IMAGE OF THE ISSUE

Otolaryngology online journal

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Volume II No1 2012

Image of the issue

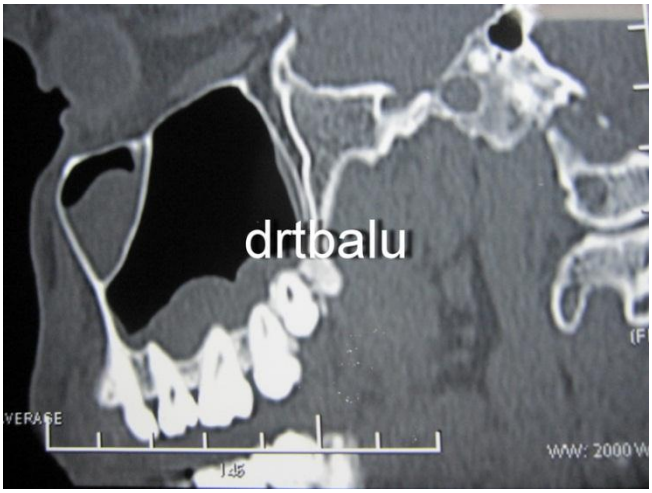
Infected Haller cell

Dr T Balasubramanian

Introduction:

Haller cells are also known as infraorbital ethmoidal cells / maxilla ethmoidal cells. These cells extend into the inferomedial portion of orbital floor. They are seen in 40% of patients.[1]

Coronal CT scan of nose and sinus showing a large Haller cell on the right side with evidence of infection



CT scan lateral view showing Haller cell below the orbit

In majority of patients Haller cells may be asymptomatic. [2]

This air cell is actually named after Albrect von Haller the Swedish Anatomist who described these air cells.

Problems caused by a Large Haller cell:

1. When infected it can cause narrowing of OMC
2. Can involve orbit
3. During Endoscopic sinus surgery it could push the natural ostium of maxillary sinus downwards and anteriorly causing difficulties during surgery
4. If this condition is not recognized preoperatively the surgeon may inadvertently enter orbit

Classification of Haller cells: [3]

Radiologically Haller cells may be classified into:

Small

Medium

Large

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December 2012

Intrinsic Rhinitis

Intrinsic Rhinitis

December 20, 2012 · Rhinology

Authors

Balasubramanian Thiagarajan

Abstract

Intrinsic rhinitis is defined as a non infective and non allergic condition characterised by nasal block, rhinorrhoea and hyposmia. This is purely a medical condition. Awareness of this condition will help us to avoid unnecessary surgical procedures on patients suffering from this disorder. Surgery should be reserved only for cases that are intractable to medical management. This article discusses the complete gamut of this disorder.

Intrinsic Rhinitis

Introduction:

Rhinitis is inflammation of nasal mucosa characterized by nasal discharge, itching and congestion. It affects 20% of the population[1] .

Intrinsic rhinitis is defined as a non infective and non allergic condition characterized by nasal block, rhinorrhoea and hyposmia. This is purely a medical condition.

Intrinsic rhinitis encompasses two separate disease entities [2]. These entities show:

1. inferior turbinate hypertrophy

2. nasal polyp formation.

symptoms

Symptoms of intrinsic rhinitis

Symptom	Eosinophilic	Non eosinophilic
Obstruction	Moderate / severe	Mild
Rhinorrhoea	Mild / Moderate	Severe
Sneezing / Pruritis	Minimal	Minimal
Hyposmia	Usual	Rare
Mucosal swelling	Marked	Mild
Inferior turbinate enlargement	Marked	Mild
Polypi	common	Never
Sinus mucosal	Common	Rare

thickening		

Clinical presentation:

Rhinitis is generally characterised by 6 main symptoms: They are

1. Congestion

2. Sneezing

3. nasal itching

4. rhinorrhoea

5. hyposmia

6. post nasal discharge

Among these main symptoms nasal itching and sneezing are features of allergic rhinitis and hence are not seen in intrinsic rhinitis. All the other symptoms are manifested in intrinsic rhinitis.

Seebohm identified two groups of patients amongst those suffering from perenial rhinitis. One group had eosinophils in their nasal secretions while the other did not have any eosinophils in their nasal secretions. Accordingly he classified intrinsic / perenial rhinitis into eosinophilic and non eosinophilic types.

Eosinophilic group: This group is characterised by marked nasal congestion, profuse rhinorrhoea, hyposmia, inferior turbinate hypertrophy and mucoid nasal secretion. Nasal polyposis frequently occurred in this group of patients.

Non eosinophilic group: In these patients nasal obstruction is very mild, rhinorrhoea is very severe. They do not have significant mucosal swelling. Inferior turbinate hypertrophy is not significant. Tendency of nasal polyp formation is rare in this group.

Aetiology of intrinsic rhinitis:

Theories regarding aetiology of intrinsic rhinitis are:

1. Autonomic imbalance

2. Airway hyperreactivity

3. Allergic reaction to unidentified allergen

4. Disturbances of Beta receptor function

Mechanisms of Beta receptor dysfunction:

1. Down regulation caused by excess endogenous noradrenaline stimualtion.

2. Down regulation and uncoupling of adenylate cyclase producedby the inflammatory mediator induced activation of protein kinase.

3. The action of Beta receptor inhibitory factor presumed to be an anti beta receptor autoantibody.

4. Dysfunction of Beta receptor kinase causing short term desensitisation of beta receptors after exposure to beta agonists.

Role of autonomic nervous system in causing intrinsic rhinitis:

The autonomic nervous system exerts its effects by secreting neurotransmitters ar their nerve

endings. The neurotransmitters secreted are adrenaline, noradrenaline, vasoactive intestinal polypeptide, acetylcholine and neuropeptide Y.

The nasal resistance to air flow is controlled by sympathetic system, whereas the nasal glands are innervated by parasympathetic nerves. Increased parasympathetic outflow causes glandular hypersecretion. Vaso active intestinal polypeptide has been known to cause this effect.The vasodilatation caused due to the effects of vaso active intestinal polypeptide is resistant to the effects of atropine.

Management:

Majority of patients with intrinsic rhinitis benefit from medical management. Only a few require

Medical management of intrinsic rhinitis:

Topical iso tonic saline spray can be used for both forms of intrinsic rhinitis. Saline spray causes a reduction of post nasal drip, sneezing and nasal congestion [3].

Topical intranasal administration of Capsaicin (derived from pepper). This irritant chemical desensitizes the sensory nerve endings of the nasal mucosa thereby reducing nasal hyperactivity [4].

Eosinophilic type:

Steroids – Topical e.g. fluticasone, budesonide. A short course of systemic steriods can be administered.

Alpha receptor agonists – Systemic e.g. pseudoephidrine Topical e.g. xylometazoline (short course)

Mast cell stabilisers – Topical cromoglycate solution.

Non eosinophlic type :

Anti cholinergic – Topical e.g. ipratropium Hyosine administered orally or as a patch.

Anti cholinergic / sympathomimetic – Imipramine orally, chlorpheniramine orally.

Surgical Management

Surgical management of Intrinsic rhinitis

Symptom	Aim	Surgery
Nasal obstruction Rhinorrohea	Turbinate reduction Turbinate resectionVidian neurectomy	Submucosal diathermy CryosurgeryLaser surgeryPartial resectionSubmucosal turbinectomyRadical turbinectomyExcision of vidian nerveEndoscopic vidian neurectomy

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June 2012

Nasal Myasis An Analysis

Nasal Myasis An Analysis

June 30, 2012 · Rhinology

Authors

Balasubramanian Thiagarajan

Abstract

This article discusses Nasal myasis, its common causes and treatment modalities followed to remove the same. Myasis Narium is also commonly known as Peenash in India. Maggots are larvae hatched from eggs laid inside the nasal cavity by certain flies. The commonest fly implicated being Sarcophagia Georginia. These flies gets attracted by stench emenating from the nasal cavity of patients. They manage to lay eggs inside the nasal cavity. These flies are capable of laying more than 200 eggs at a time. These eggs take just a day to hatch.

Nasal Myasis an Analysis

Introduction:

Myasis is defined as infestation of humans by larvae of insects. These larvae manage to feed themselves on living / dead tissues / body fluids [1]. Nasal cavity inherently is moist and has lots of fluid content. This scenario is rather common in a developing country like India. Eventhough the exact incidence of nasal myasis in India is still not known the authors crude estimate puts it somewhere at 5%. Children happen to be commonly affected because in India they run around with mucoid discharge from their nasal cavities. Overcrowding and hygenie also plays a vital role in India.

Common causes of Nasal myasis:[2]

Poorly nourished very sick patients who are unable to take care of their personal hygiene

Atrophic rhinitis – Stench from the nose attracts flies. Insensitive nasal mucosa facilitates unhindered egg laying by these flies.

Patients with leprosy affected nose

Diabetics with purulent sinus infection

Midline granulomatous lesions involving nose

Malignant lesions of nasal cavity

Nasal syphilis

In almost all these cases lack of mucosal sensation due to damage to sensory nerves supplying seems to be a predominant factor [3]in addition to the attraction of foul smell eminating from the nasal cavity.

Historically nasal myasis was used to remove necrotic tissue from wound, thus facilitating wound debridement [4].

Myasis can be classified into two types [5]:

Furuncular myasis – This is caused by larvae of flies belonging to Dermatobia Hominis. These larvae are known to penetrate the tissues forming inflammatory nodular swelling. On maturation these larvae

leave the host forming fistulous tracts.

Seconday Myasis – These are known to occur in open cavities and wounds. This type of myasis is caused by sarcophagia georgina species of flies.

Fig. 1: Midline granuloma face

Incidence[2]

True incidence levels are really not clearly known due to paucity of studies in this field. Nasal myasis commonly affects both men and women who are over the age of 50. Invariably these patients belong to lower socioeconomic class.

Symptoms:

Usually these patients present with -

Tickling sensation

Sensation of some movement within nasal cavity

Sneezing

Foul smelling blood tinged nasal discharge

Worms could be seen coming out of nasal cavity

Swollen eyelids

Nasal mucosa inflammed and odematous, may even undergo necrosis and crusting

Role of imaging:

This is essentially limited to identifying the underlying primary cause [6]for the disease, the extent of tissue destruction. This knowledge helps in formulating appropriate treatment protocol.

Prevention of nasal myasis:

This is possible only by maintaining strict hygiene.

Regular nasal douching

Regular change of clothes

Strict adherence to personal hygiene

Environmental cleanliness (prevents flies)

Treatment:

Aim of the treatment should be directed at removing all the invading maggots. This is easier said than done. Nasal endoscope 7 has facilitated this process a great deal. It helps the surgeon to visualize every nook and corner of the nasal cavity and to remove the organims under direct vision.

Fig. 2: Maggot

Topical administration of certain chemicals can make larval removal easier. They include:

Mixture of chloroform and turpentine in the ratio of 1:4

Ethylene chloride irrigation

Irrigation with a mixture of Naphtha, ether and cocaine

Ivermectin orally (one dose of 200 micrograms/kg)

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March 2013

Odontogenic cysts of upper jaw an analysis

Odontogenic cysts of upper jaw an analysis

March 27, 2013 · Rhinology

Authors

Balasubramanian Thiagarajan

Abstract

This article attempts to analyze all cases of odontogenic cysts involving upper jaw who presented at Stanley Medical college during 2007 – 2012. This article analyzes the incidence of these cysts during the above said period, age of occurence, sex prediliction if any, clinical presentations and optimal treatment modality. Common complaints with which patients presented to our Institution was swelling over jaw, next was loosening of dentition, paresthesia. 30 patients had presented with cysts involving upper jaw out of which 29 were females and one was male. All these patients underwent surgical removal of the cystic lesion.

Definition:

Odontogenic cysts are defined as epithelial cell lined cysts. This lining is derived from the odontogenic epithelium. Most of these odontogenic cysts are defined by their position than by their histology. It is important hence to describe even the site of lesion while sending the surgical specimen to a pathologist.

Introduction:

International Classification of Diseases (ICD 10) classifies odontogenic cysts involving upper jaw into:

1. Radicular cysts
2. Dentigerous cysts
3. Primordial cyst
4. Lateral periodontal cyst
5. Residual cyst
6. Odontogenic keratocyst
7. Calcifying odontogenic cyst (Gorlin cyst)
8. Globulomaxillary cyst
9. Eruption cyst

These cysts are the most common cystic lesions involving maxillofacial area [1]. Cystic lesions arecommon in the jaw bones than anywhere else in the body because of the presence of epithelial cellrests which are commonly left behind following odontogenesis.

Radicular cysts:

Synonyms – Periapical cyst, dental cyst

This is the commonest of all odontogenic cysts [2]. These cysts could also be considered as anti inflammatory cyst originating from Malassez's cell rests [3]. These cysts are caused by root infections involving roots of teeth closely related to maxillary sinus antrum. Infections / inflammation releases toxins at the apex of the tooth leading on to periapical inflammation. They stimulate the Malassez's cell rests which can be found in the periodontal ligament resulting in periapical granuloma which could either be infected or sterile. These cysts could well be sterile if the patient had received antibiotic therapy for dental infections. Radiological differentiation between granuloma and cyst could prove to be rather difficult. The general rule of the thumb being if the lesion is large in radiological imaging then it should be considered as cyst. These cysts increase in size at the expense of the surrounding bony barrier. This expansion is caused by pressure effects and effects of inflammatory enzymes over the surrounding bone. These cysts are lined by stratified squamous epithelium without keratin formation. Evidence of inflammation can be seen along the cyst wall.

Pathophysiology of Radicular cysts:

1. Inflammatory mediators / enzymes

2. Bacterial toxins

These two factors have been implicated as the probable factors contributing to Radicular cysts.

Among these two Bacterial toxins play a rather vital role. Bacterial endotoxins have been found inlarge amounts in and around necrotic tooth. These toxins have been shown to be mitogenic [4]. Theseendotoxins also stimulate expression of cytokines and chemokines [5]. Inflammatory mediators andproinflammatory cytokines released by the host tissue are known to modulate the biochemicalactivity of epidermal growth factor (EGF) there by causing increased proliferation of cellularelements. They also stimulate local fibroblasts into hyperactivity by expressing Keratinocytegrowth factor. The epithelial cell rests of Malassez are usually quiescent / stable cells. These cellsare in the G0 phase [6]of their cell cycle. These cells need to be exposed to extracellular signals topush them into the cell cycle proper. These extracellular signals are collectively known as Mitogen.

Experimentally a cell can be identified to be in the proliferative phase by their ability to expressmarkers like PCNA and Ki-67. Ki-67 marker is present in cells belonging to all phases of celldivision except G0 phase. Studies reveal increased levels of PCNA and Ki-67 markers in theepithelial lining of radicular cysts [7].

The actual binding of Mitogen (growth factor) to receptors present on the cell membrane surfaceinitiates a series of intracellular reactions pushing the cell into mitotic phase.Probable growth factors (Mitogen) involved in the pathogenesis of radicular cysts include:

1. EFG & KGF – released by stromal fibroblast

2. TGF-α – released by macrophages and lymphocytes

3. IGF (Insulin like growth factor) – released by stromal fibroblasts

In the pathophysiology of formation of radicular cysts mediators released by inflammatory cells (macrophages and lymphocyts) play a vital role [8].

Enlargement of radicular cyst:

This invariably occurs at a rather slow pace. Various factors influence the rate of expansion. Thesefactors include:

1. Mural growth

2. Hydrostatic enlargement

3. Bone resorbing factor[9]

Rapid expansion of radicular cyst is associated with increase in hydrostatic pressure within the cyst.The hydrostatic pressure within the cyst is higher than that of capillary pressure, causing fluid toenter from the capillaries into the cyst cavity. This high hydrostatic pressure within the cyst hasbeen attributed due to the amount of high molecular weight protein present in the cyst fluid. Thisprotein is released by inflammatory cells in response to inflammatory stimulus.

Role played by mast cells in radicular cyst enlargement:

Mast cells play a significant role in radicular cyst enlargement [10]. Studies reveal that there are increased number of mast cells in the subepithelial zone of these cysts. Mast cells contribute to increase in the size of these cysts in the following manner:

1. By directly releasing heparin into the lumen

2. By releasing hydrolyic enzymes

3. By releasing histamine which causes transudation of serum proteins[10]

Bone resorption by radicular cysts:

Radicular cysts causes resorption of alveolar process of maxilla. Osteoclasts have been known tocause this bone resorption. Osteoclasts need to be activated before it can reabsorb bone matrix.

Osteoclasts can be activated by:

RANKL

This reaction can be blocked by:

Osteoprotegerin (OPG)

RANKL is the molecule which activates osteoclasts by binding to its receptor RANK which isexpressed on the surface of osteoclast precursor cells, where as OPG blocks this very reactionpreventing activation of osteoclasts.

Inflammatory mediators like cytokines and Interleukins stimulate prolilferation of osteoclasts.In response to inflammation host cells are known to produce Matrix Metallo Proteinase (MMP).

This molecule is capable of degrading extracellular matrix like collagen, fibronectin and proteoglycans. Endotoxins released by bacteria also stimualtes release of MMP. This substance helps osteoclasts in the bone resorption process.

Clinical features:

As the cyst expands it causes erosion of the floor of the maxillary sinus. As soon as it enters the maxillary antrum the expansion starts to occur a little faster because there is space available for expansion. When it reaches a size wherein it fills up the whole antrum, it can erode the anterior wall

of the maxilla (in the canine fossa area). This is the weakest portion of the maxillay bone. When iterodes the anterior wall of the maxilla it could cause expansion of the maxilla which could be seen as a swelling in the cheek area. On palpation egg shell crackling may be felt in the anterior wall of the maxilla over the canine fossa. There will be associated tenderness.

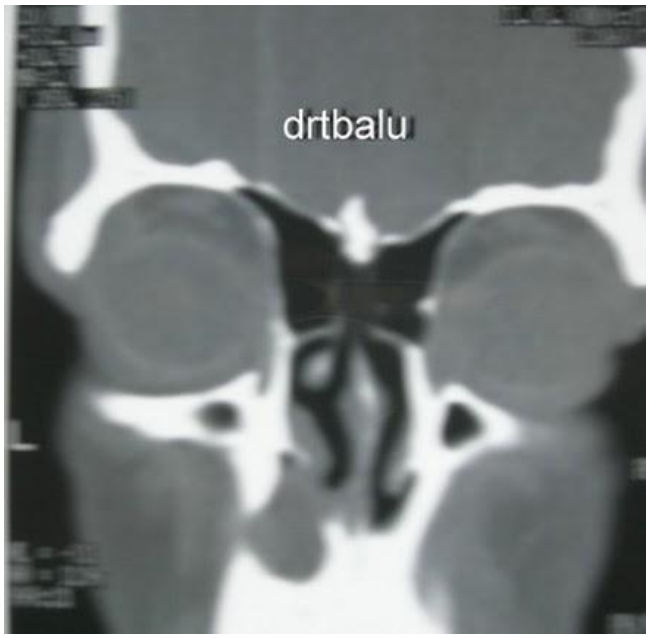
Tapping the teeth with a tongue depressor will cause tingling sensation because of involvement of the root of the teeth.

Management:

If the cyst is small, then it may resolve with endodontic therapy of the involved tooth. If the cyst is large then it will have to excised / marsupialised through Caldwell Luc approach. With the advent of nasal endoscopy, the lesion could be accessed using a nasal endoscope. The excised specimen should be sent for histopathological examination because squamous cell carcinoma could be lurking within the cystic lesion.

Clinical photo of a patient with radicular cyst

Clinical photograph showing radicular cyst maxilla being exposed via sublabial incision



Coronal CT of nose and sinuses showing radicular cyst of upper jaw

CT scan axial cut of nose and sinuses showing globulomaxillary cyst

CT scan showing dentigerous cyst with unerupted teeth

CT scan showing dentigerous cyst with unerupted teeth

Picture showing unerupted tooth within dentigerous cyst

Clinical photograph of a patient with dental cyst

Dentigerous cyst:

Also known as follicular cyst. This cyst is associated with unerupted tooth. This cyst is formed dueto accumulation of fluid between the enamel epithelium and the completely formed tooth crown.This overlying cyst prevents teeth from erupting. This cyst is almost always associated withpermanent dentition. In the upper jaw it is common in the canine tooth area. This cyst has itshighest incidence during the 2nd and 3rd decades of life.

Radiologically the presence of pericoronal radiolucency is a diagnostic pointer. This tumor shouldbe differentiated from ameloblastoma, odontogenic keratocyst and calcifying odontogenic cyst. Allthese lesions manifest with pericoronal radiolucency in routine radiographs.

Primordial cyst:

This cyst arises due to cystic changes that occur in a developing tooth bud before the actualformation of enamel and dentin matrix. Since this cyst arises from developing tooth bud the toothwould be missing from the dental arch, or if teeth are all present then the presence ofsupernumeraryteeth should be suspected.

Lateral periodontal cyst:

This cyst develops from the periodontal ligament close to the lateral surface of erupted / uneruptedteeth. This cyst is asymptomatic. The involved teeth is vital.

Residual cyst:

This cyst arises from remnants of epithelial cell rests left behind after extraction. This can alsooccur when a radicular cyst at the apex of the teeth is extracted. This cyst is commonly seen in theelderly.

Odontogenic keratocyst:

This cyst has a keratinized epithelial lining. Major draw back of this condition is its propensity to recur even after complete removal. This cyst can mimic any of the cysts described above. It needsto be identified radiologically and pathologically. This cyst is seen between wide age groups.

Calcifying odontogenic cyst (Gorlin's cyst):

This is a very rare slow growing benign tumor like cyst. This condition manifests the features of

solid mass while displaying features of tumor and cystic lesion. This cyst has equal incidence in

both maxilla and mandible.

Globulomaxillary cyst:

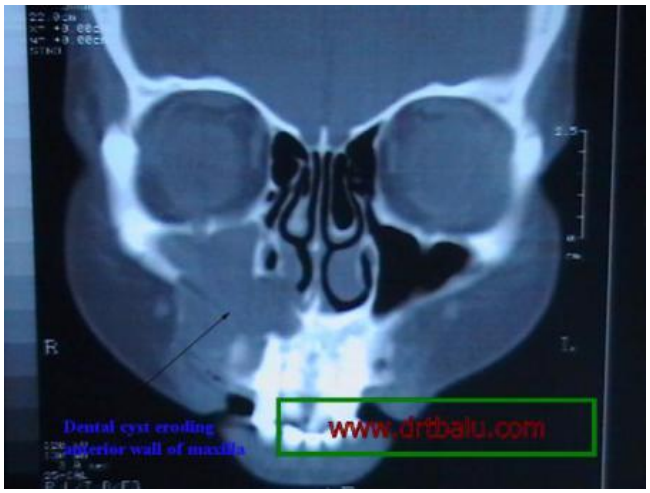
This is actually a fissural cyst arising from epithelial inclusions trapped at the line of fusion betweenthe globular portion of the median nasal process and the maxillary process. Pathologists considerthis cyst to be odontogenic rather than developmental. Radiographs show these cysts as pearshaped / circular shaped between the roots of maxillary lateral incisor and canine. Both these teethare vital in these patients.

Gingival cysts:

are of two types i.e. adult and new born. In newborn these cysts are multiple, but rarely may also be single. They are located in the alveolar ridges. In children these cysts originate from the dental lamina. They are asymptomatic and donot cause any problems. In adults these cysts are commonly found in the lower premolar area. It is usually single.

Eruption cyst:

Also known as eruption hematoma. This occurs when the erupting tooth bursts through the bone, but is yet to penetrate the overlying gingiva. Bleeding into the cyst lumen may cause discoloration giving an impression of hematoma. These cysts rupture as soon as the tooth completes eruption, hence need not be treated.



Coronal CT scan of nose and sinuses showing dental cyst right maxilla

Picture showing the site of lesion exposed prior to surgery

Figure showing Caldwel Luc procedure completed via canine fossa

Inferior meatal antrostomy being performed to facilitate drainage

Management:

Majority of odontogenic cysts can be removed surgically using sublabial incision and reaching the interior of maxillary sinus via canine fossa (Caldwel Luc procedure). It should be borne in mind that the canine fossa is the thinnest part of the maxilla and can easily be breached.After removal of the cyst via caldwel luc procedure it is mandatory to perform inferior meatal antrostomy to facilitate drainage of maxillary sinus because its mucociliary clearance mechanism is inadequate / reduced following surgery.

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March 2013

Pleomorphic adenoma hard palate a case report and literature review

Pleomorphic adenoma hard palate a case report and literature review

March 18, 2013 · *Rhinology*

Author

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Abstract

Pleomorphic adenoma is the common salivary gland tumour encountered. Most of these tumours occur in major salivary glands; Parotid gland happens to be the commonly involved one. This case report discusses a case of intraoral salivary gland tumour (in the hard palate). This case report discusses a case of pleomorphic adenoma of hard palate with a review of corresponding world literature. Even though these tumours are painless and slow growing ones, it is important to identify these cases rather early and extirpate them totally.

Introduction:

Salivary gland tumours constitute about less than 4% of all head and neck tumours. These tumours are commonly seen in adults [1] . Among the various histological varieties of salivary gland tumours pleomorphic adenoma happens to be the commonest one constituting about 70%. Most of pleomorphic adenomas involve the major salivary glands; parotid gland happens to be the common one involved. Pleomorphic adenoma is the most common tumour involving intraoral salivary glands. Palate is commonly involved, followed by lips and buccal mucosa [2] .

Most important feature of pleomorphic adenoma of minor salivary glands are:

1. There is no capsule, if present is only very thin [3] . This gives a false impression of infiltrating mass.
2. These tumours are known to cause bone erosion.

Case Report:

60 years old female patient came with;

Complaints of

1. Swelling over palate – 5 years
2. Growth slow and progressive
3. Change in voice – 3 years
4. Nasal regurgitation of fluids – 1 year
5. On and off bleeding from the mass – 6 months

On examination:

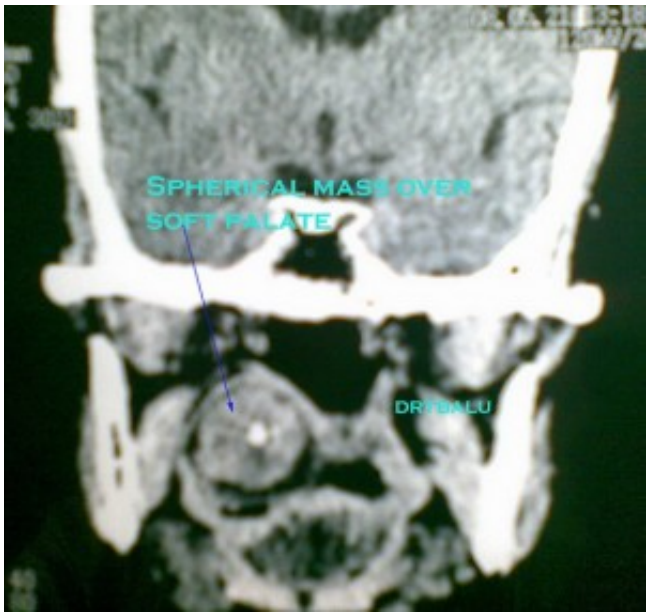
Large swelling on the right side of hard palate 3×2 inches in dimension.

Mucosa over swelling intact but for a small whitish patch? Healed ulcer.

Picture showing the palatal mass

CT scan findings:

1. Encapsulated soft tissue mass over hard palate area
2. No evidence of bone erosion
3. No evidence of intramass calcification



CT scan showing the lesion

Management:

The mass was totally excised under general anaesthesia. The patient had an uneventful post-operative period with no evidence of recurrence on follow up for 3 years.

Histopathology Report:

Pleomorphic adenoma.

Picture of gross specimen

Discussion:

Pleomorphic adenomas involving minor salivary glands are painless [4] and slow growing ones. Sometimes the growth rate could be fast. Rapid increase in size of the mass should lead to suspicion of intra lesional bleed / malignant transformation. The term pleomorphic adenoma is used to indicate the histological presence of both epithelial and mesenchymal tissues [5] .

Histological features of pleomorphic adenoma:

1. Islands of spindle cells over myxoid background
2. Inner layer of epithelial cells
3. Outer layer of myoepithelial cells

Imaging helps in ruling out palatal erosion. In all radiological images of these lesions one should look out for the presence of intact fat plane the presence of which rules out malignancy [6] .

These tumours are encapsulated and hence complete removal ensures cure. Care should be taken to leave at least 1mm margins around the lesion. While removing the mass rupture of the capsule is to be avoided to minimize recurrence [7] .

Conclusion:

Pleomorphic adenoma should be considered in all palatal tumours. Imaging helps in ruling out bone erosions in these patients. Complete extirpation of the mass is curative. One word of caution is that capsule should not be breached when attempting to surgically remove the mass because breach of capsule is associated with increasing recurrence rates.

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March 2013

Proptosis due to otolaryngology causes a study

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Proptosis due to otolaryngology causes a study

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Abstract:

Proximity of orbit to nose and paranasal sinuses makes it rather vulnerable to insults due to otolaryngological causes. The medial wall of orbit (Lamina papyracea) is rather paper thin and it happens to form the lateral wall of anterior ethmoid air cells. Infections / tumors involving paranasal sinuses can involve orbit also causing proptosis. The aim of the article is to study the common otolaryngological causes of proptosis in our Institution during 2009 – 2012.

Introduction:

Proptosis is defined as abnormal protrusion of eyeball. It has in fact been used interchangeably with exophthalmos. Purists consider exophthalmos as proptosis with lid lag (since it is commonly associated with Grave's disease). According to Epstein in Proptosis the globe protrudes out by 18 mm or less and exophthalmos is globe protrusion of more than 18 mm [1]. Henderson prefers reserving the term exophthalmos to proptosis caused by endocrine causes [2]. Proptosis should be differentiated from exorbitism which is caused by decrease in the volume of orbit causing the orbital contents to protrude anteriorly. In exorbitism proptosis is always anterior.

Anatomy of orbit and its relationships with paranasal sinuses:

Orbit is related to paranasal sinuses in two ways:

1. Anatomically by its location and by
2. 2. venous drainage (They both share the same venous drainage).

The paranasal sinuses surround the orbit from 11 o clock position superiorly to 6 o clock position inferiorly.

The orbit is pyramidal shaped formed by several bones. The superior wall of the orbit is shared by the floor of the frontal sinus, the floor of the orbit is shared by the roof of the maxillary sinus, the medial wall of the orbit is shared by the lateral wall of ethmoidal sinus. These shared bones are really thin enabling infections to travel from either direction. The medial wall of the orbit is so thin that it is termed as lamina papyracea.

Anatomical uniqueness of orbit:

1. It is a closed space

2. It is devoid of lymphatics
3. Intraorbital pressure may increase to a limit of breaking point
4. Main constituents of orbit are muscle and fat
5. Extraocular muscles are fine voluntary muscles with excessive interstitial tissue

Peculiarities of venous drainage in this area: The veins draining this area are peculiar in the following aspects:

1. The whole venous system in this area is devoid of valves, consequently a two way pathway of infection between the orbit, nasal cavity and paranasal sinuses become a reality.

2. The superior ophthalmic vein connects the facial veins to the cavernous sinus thus causing infections from face to spread to the cavernous sinus.

3. One branch of the inferior ophthalmic vein connects the orbit with that of the veins of pterygoid plexus and the other branch connects the orbit with that of the cavernous sinus.

The approximate volume of the orbital cavity is about 30 ml. Since the contents of the orbit are within a rigid confines of the orbital walls any disease process within the orbit or adjacent region has a tendency to displace the orbital contents forwards, this displacement of the globe is known as the proptosis. Other symptoms of globe displacement include diplopia, and visual loss.

Exopthalmos due to Graves disease

Proptosis due to frontoethmoidal mucocele

Methadology:

All patients who present to otolaryngology department outpatient during the period 2009 – 2012 were taken up for study. Bilateral proptosis (exopthalmos) was excluded from our study.

Type of study: Retrospective

Aim:

1. To ascertain various otolaryngological causes of proptosis
2. To ascertain the various otolaryngological clinical presentations of these patients with proptosis

All these patients under went radiological imaging to prove the exact cause for proptosis. Opthalmologist opinion was sought for all these patients to rule out ophthalmoligical cause for proptosis.

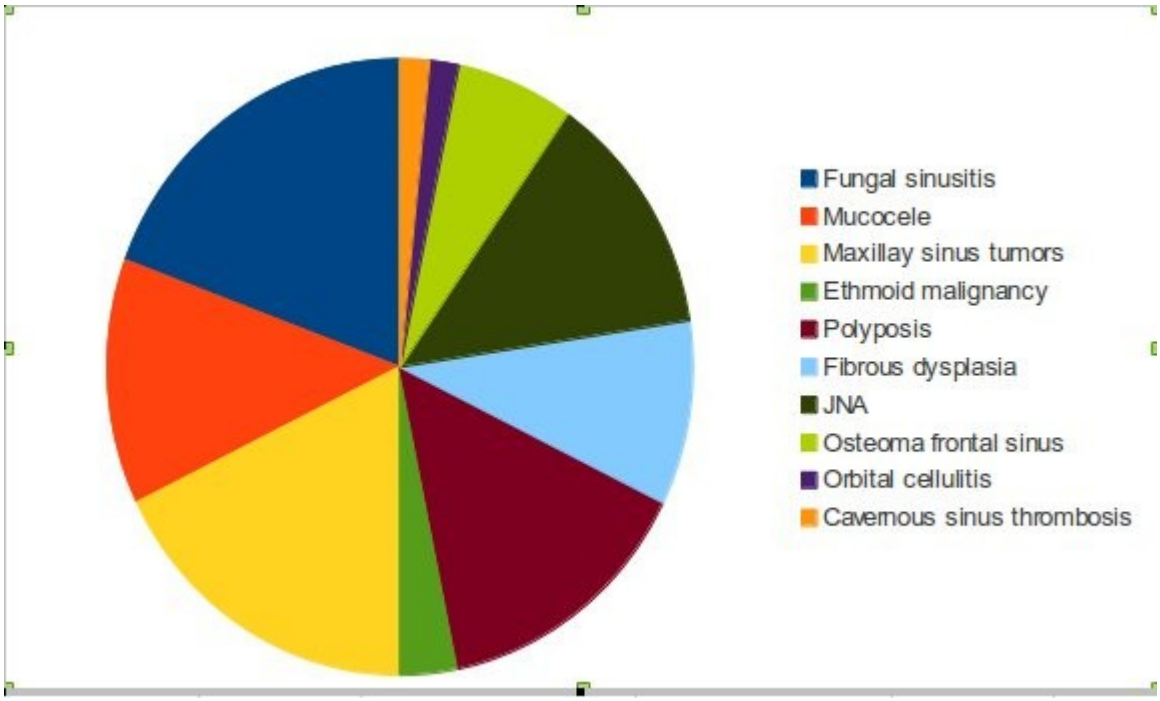
Results:

Number of patients presented to Otolaryngology department with proptosis: 62

Male – 46
Female – 16

Fungal sinusitis – 12
Fronto ethmoidal mucocele – 8
Maxillary sinus malignant tumors – 11
Ethmoidal malignancy – 2
Nasal polyposis – 9
Fibrous dysplasia [5] – 6
Juvenile nasopharyngeal angiofibroma – 8
Osteoma frontal sinus [4] – 4
Orbital cellulitis – 1
Cavernous sinus thrombosis – 1

This study reveals fungal sinusitis to be commonest otolaryngological cause for proptosis. Fungal sinusitis were initially considered as ethmoiodal polyposis causing proptosis [3].



Graph showing various otolaryngological causes of proptosis

Discussion:

This study reveals changing trends in the causative factors of proptosis[6] (This reference illustrates JNA to be the common otolaryngological cause for proptosis). Fungal sinusitis is gradually emerging as one of the common otolaryngological causes of proptosis.

The mnemonic VEIN is helpful in remembering the causes of proptosis.

V - Vascular causes

E - Endocrine causes

I - Inflammation and infective causes

N - Neoplastic causes

Imaging studies:

CT scan and MRI scan of the orbit may prove beneficial in diagnosing the cause for proptosis. The presence of fat in the orbit serves as an inherent contrast medium for the study. 3 mm cuts are ideal for the study of orbit. Ultrasound studies using either A or B mode may help in rapid diagnosis of the cause for proptosis.

Vascular causes of proptosis:

Vascular causes of proptosis can be classified into arterial and venous causes. Venous causes are due to the formation of dilated veins known as varices. Patients with these varices give a classic history of positional proptosis (proptosis varying with positions) or proptosis being induced by valsalve manuver. In patients with long standing varices there is also an associated orbital fat atrophy leading on to a transient stage of enophthalmos. In these patients a valsalva maneuver may reveal proptosis. CT scan performed with jugular venous compression or during a valsava maneuver may prove diagnostic. Surgical intervention in these patients may prove disastrous, hence observation and treatment of complications is advisable.

In dural venous sinus fistula the shunt is low flow in type and proptosis is insiduous in onset, high index of suspicion is necessary in diagnosing these patients.

Carotid cavernous fistula (high flow shunts) may arise as a result of trauma or spontaneously. These patients have subjective bruits, proptosis, chemosis and vision loss. The conjunctival vessels become arteriolised assuming a cock screw pattern. A fistula of spontaneous occurrence has a better chance of spontaneous resolution, but in intractable cases the shunt must be closed with a balloon or carotid artery ligation.

Endocrine causes of proptosis: are the most common cause of exophthalmos. The diagnosis is fairly simple because it is invariable associated with lid signs like lid lag. The major endocrine cause for proptosis is thyrotoxicosis. This condition is also known as Graves disease.

Characteristic features of endocrine causes of proptosis:

1. Presence of lid lag / lid retraction

2. Presence of temporal flare in the upper eyelid

3. Presence of orbital congestion

CT scan of the orbit show enlarged extra ocular muscles, there may also be a bulging of orbital septum due to protrusion of fat. This is pathognomonic of Grave's disease. TSH estimation show elevated levels in the serum.

Inflammatory causes of proptosis: In inflammatory proptosis the lesion could be either an idiopathic inflammatory orbital pseudotumor, or due to specific orbital inflammation. Proptosis in these

patients appear suddenly and acutely. These patients are invariably toxic and febrile. Myositis of extra ocular muscles may cause pain when eyes are being moved. There may also be associated acute dacryo adenitis. There may also be peri optic neuritis causing blindness. Orbital inflammation, perioptic neuritis and dacryo adenitis are highly responsive to oral prednisolone.

Inflammations involving the paranasal sinuses may involve the orbit causing proptosis. The interveining walls between the medial orbital wall and the ethmoidal sinuses is paper thin (lamina papyracea) which can be easily breached by infections from the ethmoidal sinuses causing spread to the orbit. In proptosis caused by ethmoidal sinus pathology the eye is pushed laterally, where as proptosis due to maxillary sinus pathology causes deviation of the eye upwards and outwards. In frontal sinus pathology the eye is deviated downwards and outwards. Commonest sinus inflammatory cause for proptosis is the formation of mucoceles in the paranasal sinuses. This commonly occur in the fronto ethmoidal regions.

Neoplastic causes of proptosis: Neoplasms involving orbit may cause proptosis. Here the eye is pushed directly forwards. This type of proptosis is known as axial proptosis. Tumors involving the optic nerve can cause axial proptosis. These patients have pain free disease. The only exception to lack of pain is patients with adenocystic carcinoma of lacrimal gland. These patients have excessive pain because the tumor infiltrates the nerves.

Neoplastic lesions involving the paranasal sinuses can also cause proptosis. The common benign tumor involving the sinuses causing proptosis are:

1. Inverted papilloma
2. Fungal infections involving the paranasal sinuses
3. Mucoceles involving the paranasal sinuses
4. Fibrous dysplasia of the maxilla
5. Osteomas involving the frontal and ethmoidal sinuses
6. Juvenile nasopharyneal angiofibroma

Coronal CT scan showing proptosis due to frontoethmoidal mucocele

Image showing proptosis due to fibrous dysplasia

Figure showing proptosis due to frontoethmoidal mucocele

Figure showing proptosis due to fungal sinusitis

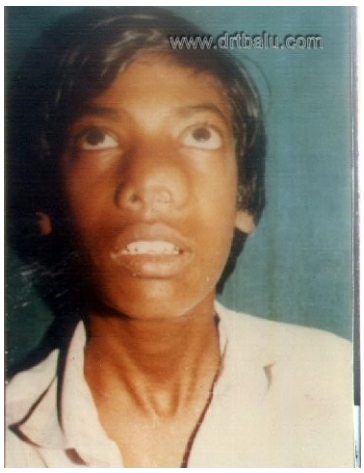


Figure showing proptosis due to JNA

Measuring proptosis using exopthalmometer:

Hertel mirror exophthalmometers are used to measure the degree of protrusion of the eyeball. The distance between the lateral orbital rim and corneal apex is used as an index for measuring proptosis. Under normal conditions this distance is roughly 18 mm, there may be individual and racial variations.

Procedure: The examiner is seated infront of the patient at the eye level of the patient. The exophthalmometer is then positioned with the blue arched support at the temporal lateral orbital walls. The instrument is maneuvered using both hands and firmly propped first against the right-hand orbital wall on the temporal side (which should be felt against the lowest part of the support point). The movable part is then set in such a way that the left-hand orbital wall lies against the lowest part of the arched support. The distance between the lateral orbital walls can then be read from the upper side of the scale; this distance can be noted for future reference. The examiner asks the patient to look straight ahead with eyelids wide open. The examiner measures for proptosis in each eye Separately by looking into the mirror (which has a millimeter scale marked on it) with one eye and moving the head horizontally until the red fixations line is at 22mm. The examiner can now determine the position of the corneal apex of the patient from the millimeter reading.

Conclusion:

This study reveals that fungal sinusitis is emerging as a common otolaryngological cause for proptosis. A large sample study will throw more light on this problem. In all the previous studies reviewed Malignant tumors involving nose and sinuses was cited to the most common otolaryngological cause for proptosis.

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August 2012

Rhinophyma

Rhinophyma

August 6, 2012 · Rhinology

Authors

Balasubramanian Thiagarajan

Abstract

This is a disfigurement of the skin lining the nose. This condition is commonly seen in elderly males. This condition is characterised by slow and progressive hypertrophy of sebaceous glands and connective tissue over the lower portion of dorsum of the nose. The term rhinophyma originates from the Greek word “Rhis” which means nose and “phyma” which means growth. This article attempts to review the hitherto published literature on this subject.

Rhinophyma

Introduction:

This condition was documented by Arab and Greek physicians way back as early as 2000 B.C[1].

These documentation have always been viewed with scepticism because those days they did nothave the wherewithal to identify this condition from the more prevalent tubeculosis, leprosy andsyphilis. This condition has been rather well documented in art and literature of yesteryears despitethe academic silence. William Shakespeare in his play William IV[2]has classically described thiscondition attributing erroneously the cause to be excessive drinking. He even coined the term BeerNose to describe this condition in detail.

Synonyms:

“Brandy Nose” “Rum Nose”. These names were christened due to erroneous presumption thatexcessive consumption of alcohol resulted in this condition.

Features:

This condition is characterised by slow progressive hypertrophy of sebaceous glands and connectivetissue over the lower portion of the dorsum of the nose. Other classic features of this probleminclude erythema, telengectasia and widened pore diameter.

Incidence:

This condition predominantly affects males in the age group of 50 – 70. Male is to female ratio isapproximately 12:1[3]. This condition is rather rare in blacks.

Etiology:

Is rather unknown. Earlier this condition used to be attributed to excessive alcohol use. This hasnot been accepted now.

This condition is considered to be an inflammatory dermatological disorder of the skincharacterised by benign hypertrophy of adnexial sebaceous structures[4]. Although etiology is stillunknown many consider it to be the end stage of Acne rosacea[5]. It was Virchow who first reportedthe correlation between acne rosacea and rhinophyma in 1846[6]. Studies have revealed a certaindegree of bacterial colonization of sebaceous glands. The role played by these colonizing microbesis not clear.Skin mite

Demodex Folliculorum has been demonstrated in these patients, exact role played bythese organim in the pathogenesis of this disease is not clear.

Vitamin deficiency, stress and increased levels of 5-alpha reductase hormone have also beenattributed to be probable cause of this problem.

Types of rhinophyma:

1. Glandular

2. Fibrous

3. Fibroangiomatous

Glandular variety:

In these patients nasal enlargement is due to marked lobular hyperplasia of sebaceous glands.

Fibrous variety:

Overgrowth of skin over the nose is due to increase in the density of nasal connective tissueassociated with variable amount of sebaceous gland hyperplasia.

Fibroangiomatous variety:

The nose in this particular variety is strikingly reddish and coppery in nature. This is due toenlarged and edematous connective tissue with enlarged veins.

Gross morphological features of rhinopyma:

Telangiectasia

Hypervascularity – Is responsibe for facial flushing[7].

Thickened nasal cutaneous layer

Nodularity over nasal cutaneous layer covered with atrophic skin. Pores appear large andexpandedProminent cutaneous follicles from which foul smelling sebum can be expressed

Histopathology:

It is always better to confirm the diagnosis histopathologically as this condition could be mimickedby angiosarcoma, basal cell carcinoma, tuberculosis, leprosy, syphilis and sarcoidosis.

Histopathological features include:

1. Sebaceous gland hyperplasia

2. Connective tissue hyperplasia

3. Signs of chronic inflammation

4. Dilated blood vessels

Treatment:

Medical management:

This has proved to be highly disappointing[8]. None of the so called medical management modalityhas shown promise. Still in early lesions it is worth while trying them out. These include:

1. Oral Tetracycline

2. Topical metronidazole

3. Topical Tretinoin

Clinical photograph of a patient with Rhinophyma

Retinols can be used to reduce the volume of sebaceous tissue.Calcineurin inhibitors like tacrolimus ointment have also been used in early phases.

Surgical Management:

This is considered to be a gold standard in the management of rhinophyma. It should be consideredin all patients with moderate to large lesions. The aim of surgery is to remove hypertrophied tissueand normalization of nasal contour.

Surgeries include:

1. Total excision with skin transplant of the defect produced

2. Partial excision and healing by second intention

3. Laser ablation

4. Cryosurgery

5. RF cautery

6. Coablation

7. Using microdebriders

8. Dermabrasion

9. Laser surgery

Prior to surgery the following precautions need to be taken:

1. Avoidance of alcoholic beverages 1 week prior to surgery

2. Antiinflammatory drugs to be stopped one week prior to surgery.

3. Running nose / nasal allergy should be aggressively treated prior to surgery

4. Smoking should be stopped 3 months prior to surgery. Smoking can cause a reduction in thevascularity compromising flap

Conventional surgical procedures include:

Total excision with flap cover of the lesion, or partial excision of the lesion allowing basal skinappendages to re-epithelize [9].

Use of Shaw scalpel:

Shaw scalpel (this was designed by Robert Shaw) can be used to reduce / shave hyperplastic tissue.This scalpel can be used as cauterizing device. 10 blade is used with a temperature setting of 150degrees centigrade. The tissue is actually removed in layers as thin strips. Bleeders are sealedinstantly by the hot scalpel blade.

Features of Shaw Scalpel:

It has disposable blades which comes in two standard sizes No. 10 and No. 15. These blades aremade of surgical steel and coated with copper (for better electrical conductivity). It has an outercoating of Teflon which permits easy removal of clotted blood. A controller unit provides pulseddirect current which heats the scalpel blade between 110-270 o C. There is facility in the controlunit to vary the temperature of the scalpel blade in 10o C increments.

Shaw scalpel

Use of laser:

Carbondioxide laser has been used in the management of rhinophyma. Using this instrumentenables the surgeon to maintain haemostasis throughout the surgical procedure. It is also easy tocontinuously monitor the depth of surgical excision [10]. NdYag laser has also been used with greatdegree of success.

Use of Cryo surgery:

Cryosurgery using liquid nitrogen can be used to debulk these lesions.

Use of coablator:

Coablator can be used to remove excess tissue from the nasal dorsum. It also has the advantage ofsimultaneous sealing of bleeders.

Radiofrequency surgery:

Radiofrequency surgery [11]using loop electrodes has shown immense promise in debulking of thelesion.

Use of tissue glue:

This can be used to reduce bleeding from the denuded area.

Post operatively ointment can be appplied over the wound. Dressing need not be applied.

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February 2012

Rhinosporidiosis

Rhinosporidiosis

A review

February 23, 2012 · Rhinology

Authors

Balasubramanian Thiagarajan

Definition:

Rhinosporidiosis has been defined as a chronic granulomatous disease characterised by production of polyps and other manifestations of hyperplasia of nasal mucosa. The etiological agent is Rhinosporidium seeberi.[1]

Rhinosporidium seeberi: was initially believed to be a sporozoan, but it is now considered to be a fungus and has been provisionally placed under the family Olipidiaceae,[5] order chritridiales of phycomyetes by Ashworth. More recent classification puts it under DRIP'S clade. Even after extensive studies there is no consensus on where Rhinosporidium must be placed in the Taxonomic classification. It has not been possible to demonstrate fungal proteins in Rhinosporidium even after performing sensitive tests like Polymerase chain reactions[2] .

History[3]:

1892 – Malbran observed the organism in nasal polyp

1900 – Seeber described the organism

1903 – O'Kineley described its histology

1905 – Minchin & Fantham studied O'Kineley's tissue and named the organism as Rhinosporidium Kinealyi

1913 – ZSchokke reported similar organism in horses and named it Rhinosporidium equi

1923 – Ashworth described its life cycle[4]

1924 – Forsyth described skin lesion

1924 – Thirumoorthy reported the first female patient

1936 – Cefferi estabilised the identity of R. Seeberi and R. Equi

1953 – Demellow described the mode of its transmission

Incidence and Geographical distribution:

Of all the reported cases 95 % were from India and Srilanka. An all India survey conducted in 1957 revealed that this disease is unknown in states of Jummu & Kashmir, Himachal pradesh, Punjab, Haryana, and North Eastern states of India. In the state of TamilNadu 4 endemic areas[8] have been identified in the survey, (Madurai, Ramnad, Rajapalayam, and Sivaganga). The common denominator in these areas is the habit of people taking bath in common ponds.

Theories of mode of spread:

1. Demellow's theory of direct transmission[6]

2. Autoinoculation theory of Karunarathnae (responsible for satellite lesions)[7]

3. Haematogenous spread – to distant sites

4. Lymphatic spread – causing lymphadenitis (rarity)

Demellow's theory of direct transmission – This theory propounded by Demellow had its acceptance for quite sometime. He postulated that infection always occured as a result of direct transmission of the organsim. When nasal mucosa comes into contact with infected material while bathing in common ponds, infection found its way into the nasal mucosa.

Karunarathnae accounted for satellite lesions in skin and conjunctival mucosa as a result of auto inoculation.

Rhinosporidiosis affecting distant sites could be accounted for only through haematogenous spread.

Karunarathnae also postulated that Rhinosporidium existed in a dimorphic state. It existed as a saprophyte in soil and water and it took a yeast form when it reached inside the tissues. This dimorphic capability helped it to survive hostile environments for a long period of time.

Reasons for endemicity of Rhinosporidiosis[2]:

It has to be explained why this disease is endemic in certain parts of South India and in the dry zone of Srilanka. If stagnant water could be the reason then the chemical and physical characteristics of the water needs to be defined. In addition other aquatic organisms may also be playing an important synergistic reaction. This aspect need to be elucidated. Text book of microbilogy is repleate with examples of such synergism i.e. lactobacillus with trichomonas, and Wolbachia with filarial nematodes.

Host factors responsible for endemicity: Eventhough quite a large number of people living in the endemic areas take bath in common ponds only a few develop the disease. This indicates a predisposing, though obscure factors in the host. Blood group studies indicate that rhinosporidiosis is common in patient's with group O (70%), the next high incidence was in group AB. Jain reported that blood group distribution is too variable to draw any conclusion. Larger series must be studied for any meaningful analysis. HLA typing also must be studied. The possibility of non-specific immune reactivity especially macrophages in protecting the individual from Rhinosporidium seeberi must be considered.

Life cycle: (Ashworth) Spore is the ultimate infecting unit[4]. It measures about 7 microns, about the size of a red cell. It is also known as a spherule. It has a clear cytoplasm with 15 – 20 vacuoles filled with food matter. It is enclosed in a chitinous membrane. This membrane protects the spore from hostile environment. It is found only in connective tissue spaces and is rarely intracellular.

Life cycle of Rhinosporidium seeberi (Old)

The spore increases in size, and when it reaches 50 – 60 microns in size granules starts to appear, its

nucleus prepares for cell division. Mitosis occurs and 4, 8, 16, 32 and 64 nuclei are formed. By the time 7th division occurs it becomes 100 microns in size. A fully mature sporangia measures 150 – 250 microns. Mature spores are found at the centre and immature spores are found in the periphery. The full cycle is completed within the human body.

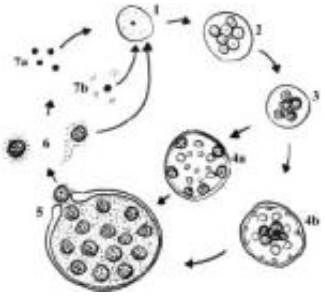
Life cycle (recent): Since rhinosporidium seeberi has defied all efforts to culture it, any detail regarding its life cycle will have to be taken with a pinch of salt. This life cycle has been postulated by studying the various forms of rhinosporidium seen in infected tissue.

Trophozoite / Juvenile sporangium – It is 6 – 100 microns in diameter, unilamellar, stains positive with PAS, it has a single large nucleus, (6micron stage), or multiple nuclei (100 microns stage), lipid granules are present.

Intermediate sporangium – 100 – 150 microns in diameter. It has a bilamellar wall, outer chitinous and inner cellulose. It contains mucin. There is no organised nucleus, lipid globules are seen. Immature spores are seen within the cytoplasm. There are no mature spores.

Mature sporangium – 100 – 400 microns in diameter, with a thin bilamellar cell wall. Inside the cytoplasm immature and mature spores are seen. They are found embedded in a mucoid matrix. Electron dense bodies are seen in the cytoplasm. The bilamellar cell wall has one weak spot known as the operculum. Maturation of spores occur in both centrifugal and centripetal fashion. This spot does not have chitinous lining, but is lined only by a cellulose wall. The mature spores find their way out through this operculum on rupture. The mature spores on rupture are surrounded by mucoid matrix giving it a comet appearance. It is hence known as the comet of Beattee

Mature spores give rise to electron dense bodies which are the ultimate infective unit.



Life cycle of Rhinosporidium Seeberi (New)

1 – Trophozoite (juvenile sporangium)

2 & 3 – Immature bilamellar sporangia

4a & 4b – intermediate sporangia with centrifugal and centripetal maturation of endospores

5 – Mature sporangium with spores exiting through the operculum

6 – Free endospore with residual mucoid material giving it a comet like apperance (comet of Beattie)

7a – Free electron body (ultimate infective unit)

7b – Free electron dense body surrounded by other electron dense bodies which are nutritive granules

Clincial classification of Rhinosporidiosis:

1. Nasal

2. Nasopharyngeal

3. Mixed

4. Bizzarre (ocular and genital)

5. Malignant rhinosporidiosis (cutaneous rhinosporidiosis)

Common sites affected:

Nose – 78%

Nasopharynx – 68%

Tonsil – 3%

Eye – 1%

Skin – very rare

Fig. 1: Oropharyngeal rhinosporidiosis

Fig. 2: Nasopharyngeal rhinosporidiosis

Gross features of rhinosporidiosis:

Lesions in the nose can be polypoidal, reddish and granular masses. They could be multiple pedunculated and friable. They are highly vascular and bleed easily. Their surface is studded with whitish dots (sporangia). They can be clearly seen with a hand lens. The whole mass is covered by mucoid secretion. The rhinosporidium in the nose is restricted to the nasal mucous membrane and does not cross the muco cutaneous barrier.

Histopathology of nasal rhinosporidiosis:

There is papillomatous hyperplasia of nasal mucous membrane with rugae formation. The epithelium

over the sporangia is thinned out, foreign body giant cells can be seen. Accumulation of mucous in the crypts seen with increased vascularity. The increased vascularity is responsible for excessive bleeding during surgery. Increased vascularity is due to the release of angiognenesis factor from the rhinosporidial mass. Rhinosporidial spores stain with sudan black, Bromphenol blue etc.

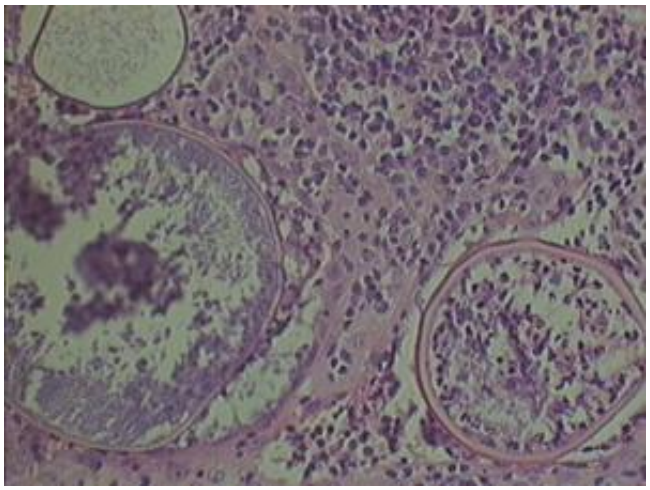


Fig. 3: Rhinosporidiosis Histopathology

Endosporulation[2]:

Endospores represent asexual spores of Rhinosporidium seeberi. After nuclear division in the juvenile sporangia, endospores are forrmed by condensation of cytoplasm around the nuclei with the formation of cell walls. This process is known as endosporulation. These endospores have been postulated to develop from the inner sporangial wall. Endospores are liberated from the sporangium by bing shot out from the sporangium after its rupture (as suggested by Beattee), or through the operculum as suggested by Ashworth, or by osmotic mechanism as suggested by Demello. Endospores are thick walled measuring about 7 microns in diameter, round in shape and stains with PAS. It has a vesicular nucleus and a granular cytoplasm. The peripheral cytoplasm is vacuolated containing deeply staining bodies called as spherules. These bodies give the spore a morullated appearance and hence the term spore morullae.

Features of rhinosporidiosis:

The cardinal features of rhinosporidiosis are:

1. chronicity

2. recurrence and

3. dissemination.

The reasons for chronicity are

1. Antigen sequestration – The chitinous wall and thick cellulose inner wall surrounding the endospores is impervious to the exit of endosporal antigens from inside, and is also impermeable to immune destruction. However this sequestered antigen may be released after phagocytosis.

2. Antigenic variation – Rhinosporidial spores express varying antigens thereby confusing the whole immune system of the body.

3. Immune suppression – ? possible release of immuno suppressor agents

4. Immune distraction – Studies of immune cell infiltration pattern have shown that immune cell infiltration has occurred in areas where there are no spores, suggesting that these infiltrates reached

the area in response to free antigen released by the spores. This serves as a distraction.

5. Immune deviation

6. Binding of host immunoglobins

Treatment:

Surgery is the treatment of choice. Rhinosporidial mass can be removed intranasally, the only problem being bleeding. Post operatively the patient is started on T. Dapsone[9] in dose of 100 mg / day for a period of 6 months.

Unsolved problems:

Habitat – Breeds in ponds (highly theoretical, spores have not been isolated from ponds even on intense effort)

Lifecycle – In the absence of viable ways to culture the organism the life cycle remains highly speculative

Pathogenicity – does not fullfill any of the 4 criterial laid down by Koch regarding the infectivity

Morphology

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November 2012

Role of x-rays in Rhinology

Role of x-rays in Rhinology

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Abstract:

X-rays in the present day context is considered to be outdated by Rhinologists. CT scan images have replaced conventional x-ray imaging. Current concensus is that still x-rays have a role in the field of rhinology. Its inherent advantages like easy availability, cost effectiveness makes this investigation still relevant in the present day scenario. Bony lesions involving nose and sinuses, can be evalutated with reasonable degree of accuracy by performing conventional radiographs. Air present inside the paranasal sinuses serve as excellent contrast medium for plain x-ray evaluation. Pathologies involving paranasal sinuses encroach upon these air spaces causing alterations in their translucency.

Introduction:

With the advent of CT / MRI imaging plain radiographs are losing their relevance as far as rhinological diagnosis is concerned. This article attempts to explore the currently prevailing indications for the use of plain radiographs in rhinology. Eventhough CT scan provides improved resolution of soft tissue densities it has its own draw backs like increased cost of investigation and not so easy availability. Paranasal sinuses are air filled cavities. Pathology involving these structures cause varying degrees of attenuation of these air contrast medium which can easily be observed in any conventional radiographs. Certain pathological conditions can cause accumulation of fluid within the sinus cavity. This can be clearly demonstrated if plain x-rays are taken with patient in erect position. Fluid levels can clearly be demonstrated even in plain radiographs.

Advantages of x-ray imaging in rhinology include:

1. Cost effectiveness of the investigation
2. Easy availability
3. Currently available digital x-ray imaging techniques provide better soft tissue and bone resolution when compared to conventional x-rays

Disadvantages of conventional radiographs:

1. Plain radiographs have a false positive rate of 4% [1].

2. Plain radiographs have false negative rate of more than 30% [2].
3. Difficulties in patient positioning

Standard radiograph positions for studying nose and paranasal sinuses:

Radiographic study of paranasal sinuses are standardized around three positions. These include:

1. Two anatomical positions – Coronal and saggital

2. Radiographic – This position pertains to radiographic baseline. This is actually an imaginary line drawn from the outer canthus of the eye to the mid point of external auditory canal.

Positions used to study paranasal sinuses include:

1. Occipito mental view (water's view)

2. Occipital frontal view (Caldwell view)

3. Submento-vertical view (Hirtz view)

Water's view:
Also known as occipito mental view is the commonest view taken. This view was developed by Waters and Waldron in 1915. This was actually a modification of occipito frontal projection (Caldwell view) [3]. Patient is made to sit facing the radiographic base line tilted to an angle of 45 degrees to the horizontal making the sagittal plane vertical. The radiological beam is horizontal and is centered over a point 1 inch above the external occipital protruberance. In obese patients with a short neck it is virtually impossible to obtain an angulation of 45 degrees. These patients must be made to extend the neck as much as possible and the xray tube is tilted to compensate for the difference in angulation. The mouth is kept open and the sphenoid sinus will be visible through the open mouth. If the radiograph is obtained in a correct position the skull shows a foreshortened view of the maxillary sinuses, with the petrous apex bone lying just beneath the floor of the maxillary antrum. In this view the maxillary sinuses, frontal sinuses and anterior ethmoidal sinuses are seen. The sphenoid sinus can be seen through the open mouth.

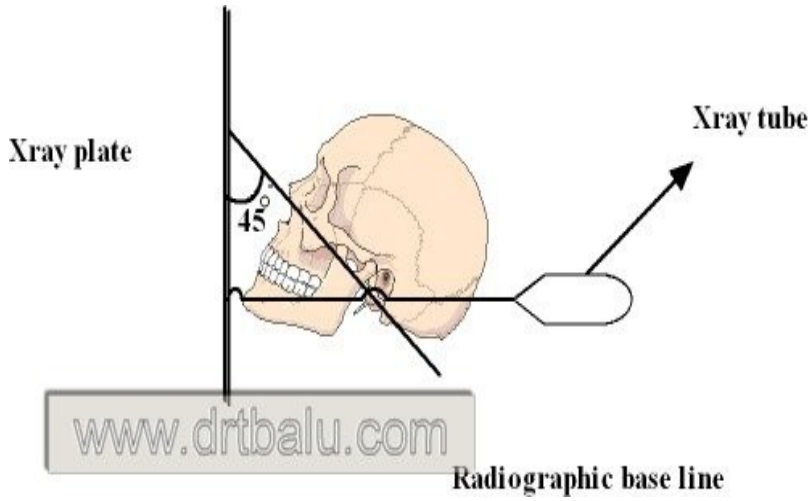


Diagram showing the position of the skull while taking x-ray sinus water's view

If the antrum in water's view demonstrates a loss of translucency which could be an indicator of fluid level, then another x ray is taken with a tilt of saggital plane to an angle of 30 degrees. This view will clearly demonstrate movement of fluid to a new position. In this view the fluid moves towards the lateral portion of the antrum where it can clearly be seen.

In x-ray pararanasal sinuses water's view chronic maxillary sinusitis is displayed as:

1. Clouding of maxillary sinuses
2. Opacification of maxillary sinuses
3. Mucosal thickening of maxillary sinuses
4. Fluid level seen in the maxillary sinuses

Among these 4 signs least accurate happens to be clouding of maxillary sinuses. This has a false positive value of more than 30% in some studies. Total opacification of maxillary sinus is a more reliable sign of infection with accuracy levels ranging between 80-92%[4].
Sources for error in interpreting xray paranasal sinuses waters view could be:

1. Anatomical
2. Technical

Anatomical causes for interpretation errors in xray paranasal sinuses water's view:

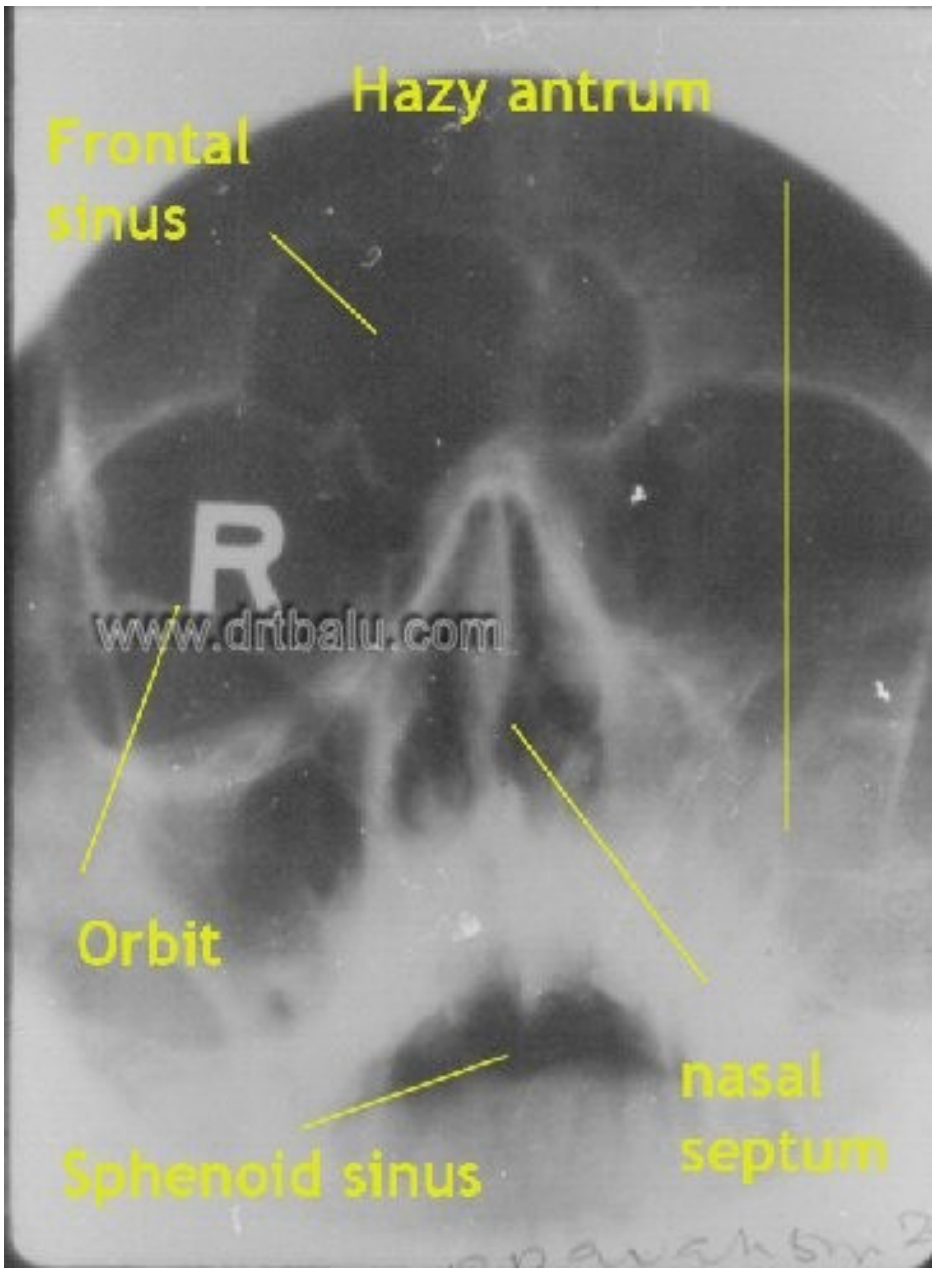
1. Hypoplastic sinus – This could cause clouding / total opacification of maxillary sinus
2. Orbital floor exposure- This could appear as false mucosal thickening
3. Zygomatic recess – This could appear as false mucosal thickening
4. Superior orbital fissure- This could appear as false mucosal thickening or false interpretation as fluid level.

Technical causes for interpretation errors in x-ray paranasal sinuses water's view include:

1. Soft exposure – This can cause an impression of clouding of maxillary sinuses
2. Lateralization of radiographic tube – This will cause mucosal thickening on the side of lateralization to be missed in the radiograph.
3. Inadequate tilting of tube / head – This will cause an artificial impression of presence of fluid level.
4. Marked tilting of tube / head – This will cause impression of clouding of maxillary sinuses
5. Patient in recumbent postion – This can cause retained secretions in the maxillary sinuses to be missed.

X-ray paranasal sinuses Water's view clearly shows:

1. Maxillary sinuses
2. Frontal sinus
3. Ethmoidal sinuses
4. Orbit
5. Sphenoid sinus
6. Nasal cavities
7. Alveolar arches
9. Xygoma



X-ray paranasal sinuses Water's view showing different structures and a hazy maxillary sinus on the left side

X-ray paranasal sinuses water's view is still a viable investigation in patients with facio maxillary trauma. Examination of Dolan's line in x-ray sinuses water's view helps in identifying patients with zygomatico-maxillary complex fractures.

Dolan's line:

This was first popularized by Dolan etal. They described three lines which in conjunction resembled elephant's head in profile.

1. Orbital line – This resembles the elephant's ear. Disruption of this line could be

due to fracture through lateral orbital rim or diastasis of fronto-zygomatic suture line. Disruption of inferior portion of orbital line could be due to fracture involving inferior rim of orbit / floor of orbit.

2. Zygomatic line – This line resembles elephant's forehead and trunk. This line follows the lateral orbital rim to the superior margin of zygomatic arch. Disruption of this line indicates fracture involving zygoma.

3. Maxillary line – This resembles elephant's chin and trunk. This line follows the lateral wall of the maxillary sinus to the inferior margin of zygomatic arch. Fractures are expected in this zone.

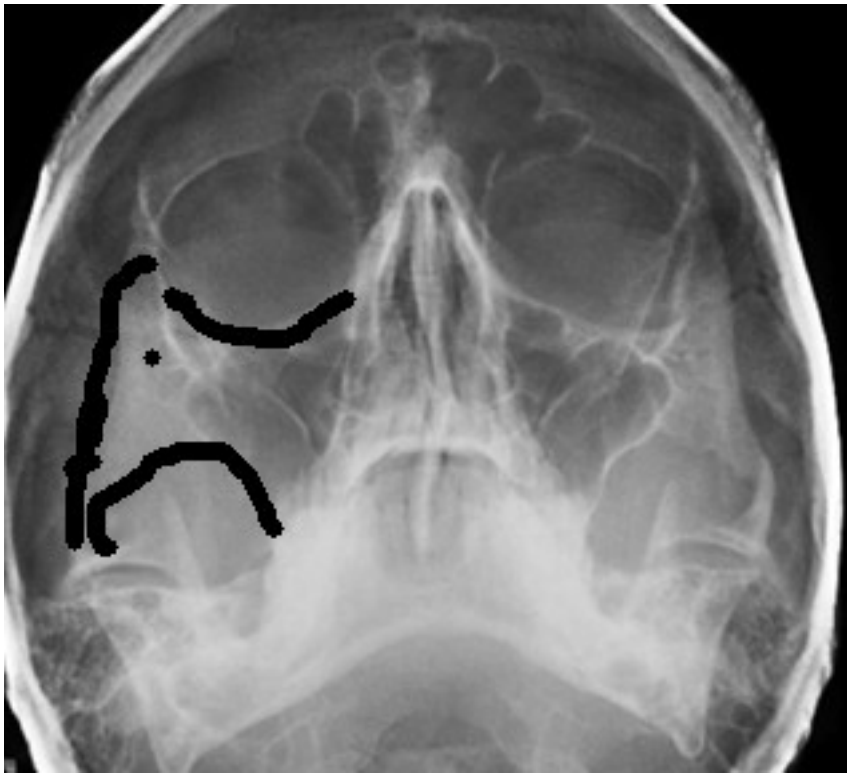
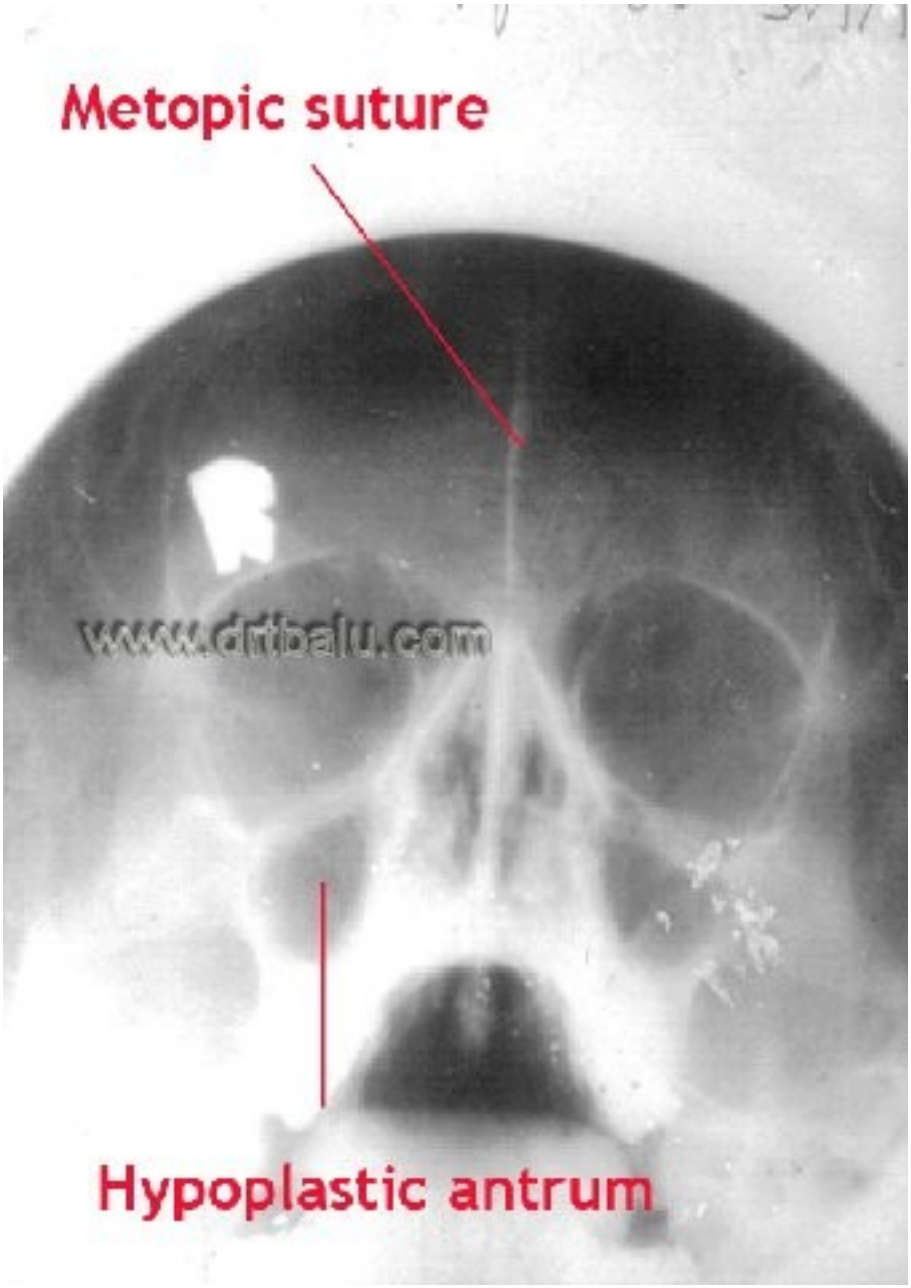


Image showing Dolan's line marked in x-ray sinuses Water's view

In x ray para nasal sinuses waters view the normal frontal sinus margins show scalloping. Loss of this scalloping is a classic feature of frontal mucocele. If frontal sinus is congenitally absent (agenisis) then a suture line known as the metopic suture is visible in the fore head area. Sometimes a pair of large anterior ethmoidal air cells may take up the place of frontal sinuses. Here too the metopic suture line is visible. This suture divides the two halfs of frontal bone of the skull in infants and children. This suture line usually disappears at the age of 6 when it fuses. If this suture is not present at birth it will cause a keel shaped deformity of the skull (trigonocephaly).



X-ray sinuses water's view showing Metopic suture with hypoplastic maxillary sinus and absent frontal sinus

Since hypoplastic antra are associated with sclerosis of its margins, it will be very difficult to perforate the medial wall of the antrum while performing antrostomy.

In conditions like malignancy involving the maxillary antrum X ray sinuses waters view shows the following features:

Expansion

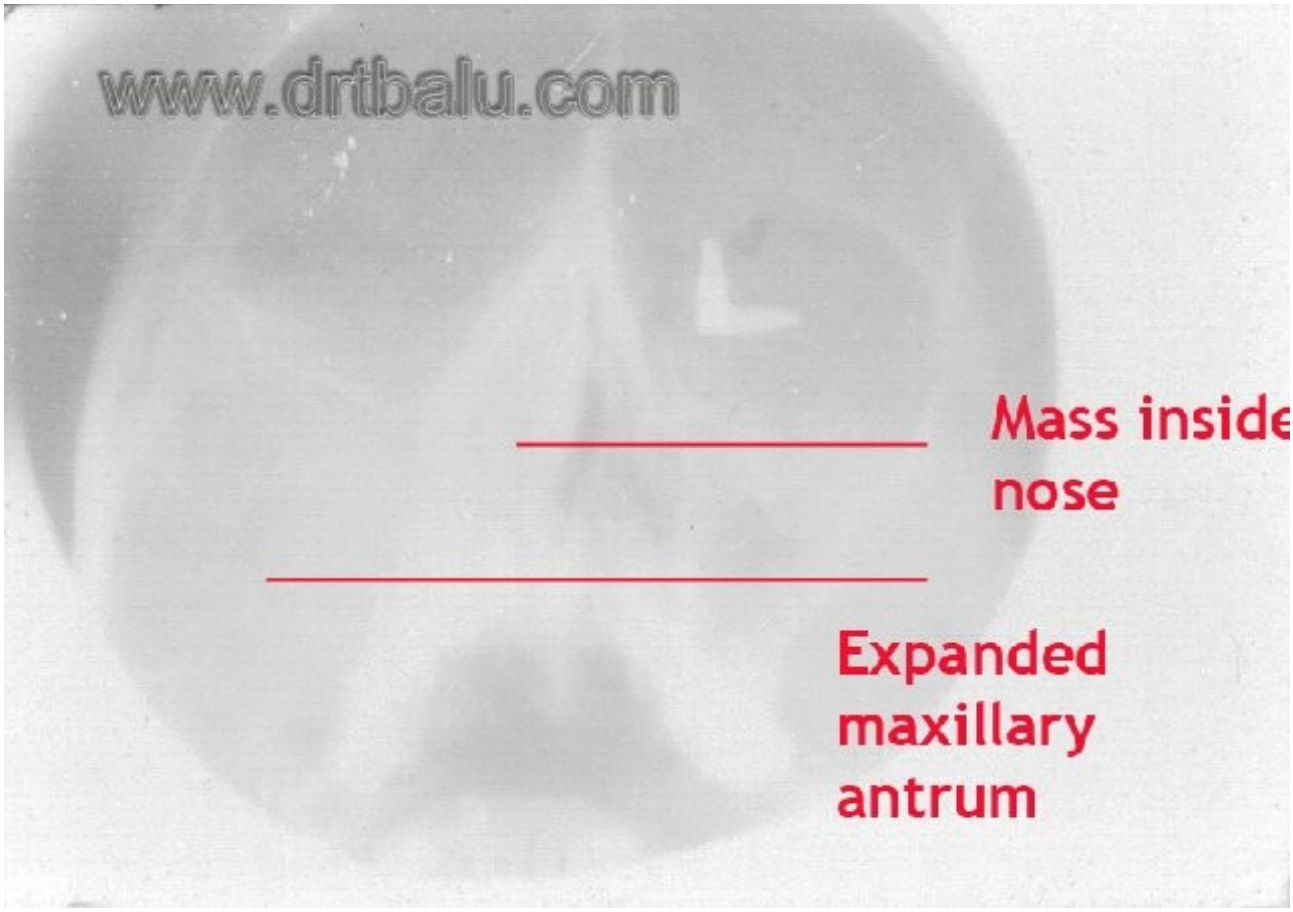
Erosion

Opacity.

Expansion is characterised by increase in the size of maxillary antrum when compared to its counter part on the opposite side.

Erosion may occur in the medial wall of the antrum or in its antero lateral wall. The canine fossa area is the thinnest portion of maxillary antrum antero lateral wall. Erosion is hence common in this area.

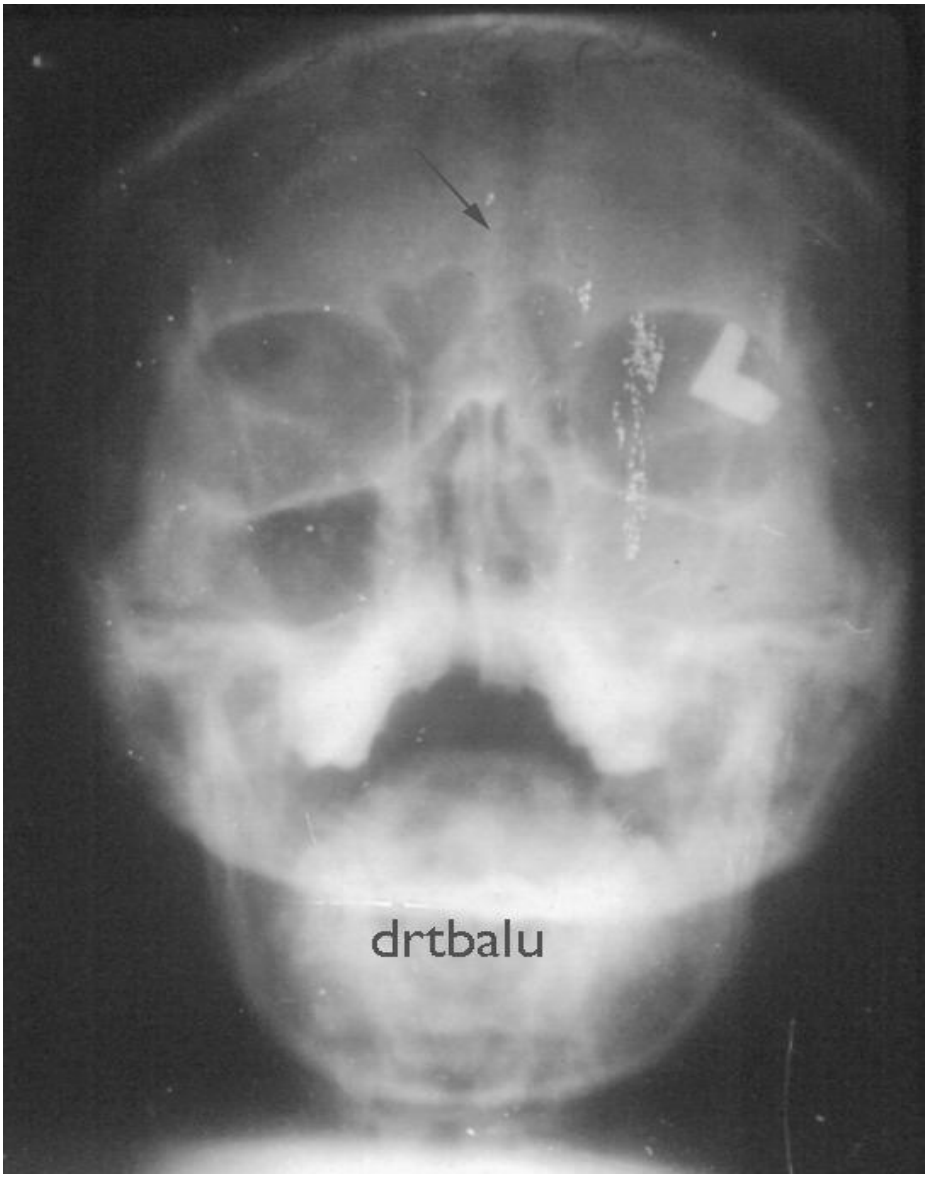
Opacity is the term used to describe a maxillary sinus antra involved with malignant growth. This opacity is due to the periosteal reaction due to malignant growth.



X-ray sinuses water's view showing expansion and erosion of right maxillary antrum due to malignancy

According to Veterans Affairs general medicine clinic study [5] using the following criteria to diagnose sinusitis from x-ray sinuses water's view improved its accuracy to more than 80%. These criteria include:

1. Presence of air fluid level
2. Sinus opacity
3. Mucosal thickening geater than 6 mm



X-ray PNS water's view shows metopic suture (arrow) which is a persistent frontal suture.
Usually it disappears at the age of 6 with the normal development of frontal sinuses. Persistence of Metopic suture (i.e. sutura frontalis persistens). In patients with agenesis of frontal sinuses the metopic suture is still persistent. In the above x-ray the frontal sinus is absent and is replaced by a pair of large anterior ethmoidal air cell. (Note: The left maxillary sinus appear hazy, possibly due to sinusitis).*

Routine CT scan imaging is not indictated routinely to diagnose patients with acute sinusitis. Eventhough it has high specificity in diagnosing acute sinusitis it has low specificity. Studies reveal that more than 40% of asymptomatic patients too show CT scan abormalities indicating presence of sinusitis [6].

Digital x-rays:

With the advent of digital imaging techniques the cost of imaging has drastically reduced. The quality of images generated are much better than analog ones which was captured on a film. In addition this procedure involves less amount of radiation to the patient when compared to that of conventional x-ray techniques.

Occipito-frontal view (Caldwell view):

This position is ideally suited for studying frontal sinuses. In this position the frontal sinuses are in direct contact with the film hence there is no chance for any distortion or geometric blur to occur. Hence it is ideally suited to create frontal sinus templates for external surgeries involving frontal sinuses.

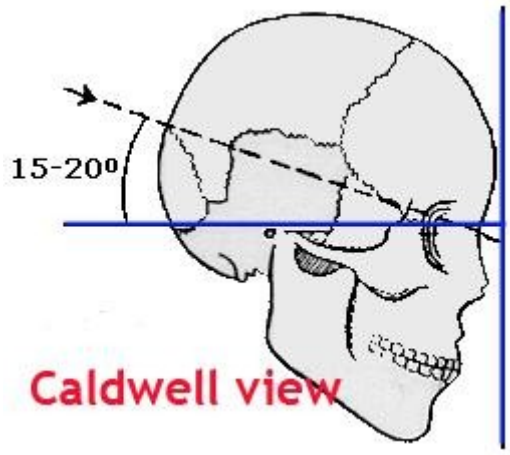
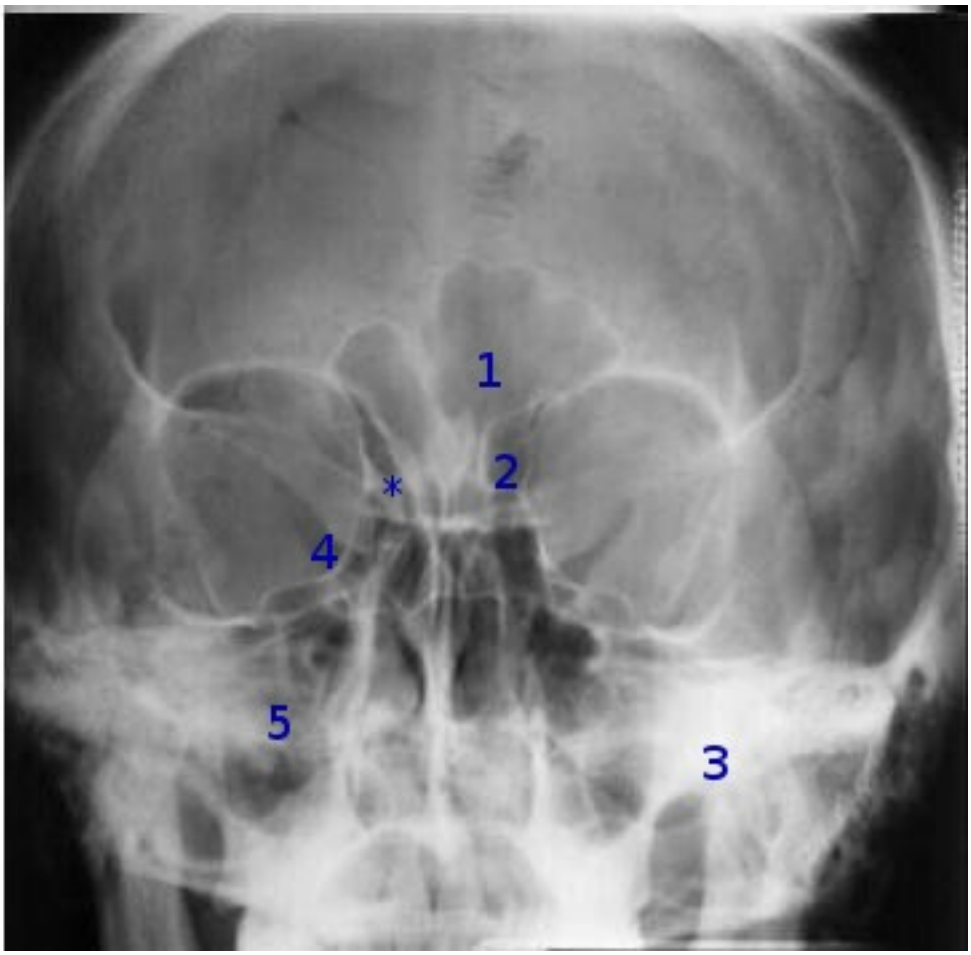


Figure showing patient position for Caldwell view radiograph

To get a Caldwell view the patient is made to sit in front of the film with the radiographic vbase line tilted to an angle of 15 - 20 degrees upwards. The incident beam is horizontal and is centered 1/2 inch below the external occipital protruberance. This view is also known as the frontal sinus view. In this view the petrous portion of the temporal bone obscures the visualization of maxillary sinuses. Frontal sinuses can be clearly studied using this view. This view also helps in templating the frontal sinus before external Fronto ethmoidectomy surgeries. This view also clearly demonstrates frontal and ethmoidal sinuses. Maxillary sinus is not clearly visible because the petrous apex obscures its view.

In this view a small indentation / groove can be seen along the upper portion of the medial wall of orbit. This groove is caused by anterior ethmoidal artery and nasociliary nerve. The ethmoido-maxillary plate is clearly seen in this view. This is actually a useful landmark for localizing spread of tumors.



X-ray sinuses Caldwell view:
1- Frontal sinus
2- Ethmoid sinus
3-Petrous apex
4-Inferior orbital fissure
5-Maxillary sinus obscured by petrous apex
*- Site of anterior ethmoidal artery

Submentovertical view (Hritz view):

This view is primarily taken to view sphenoid sinus. Fluid levels in sphenoid sinuses are clearly shown in this view. To take an x ray in this position, the back of the patient is arched as far as possible so that the base of skull is parallel to the film. The x ray beam is centered in the midline at a point between the angles of the jaws. In elderly patients this view can be easier to achieve if carried out in the supine position with the head hanging back over the end of the table. This view also demonstrates the relative thicknesses of the bony walls of the antrum and the frontal sinuses.

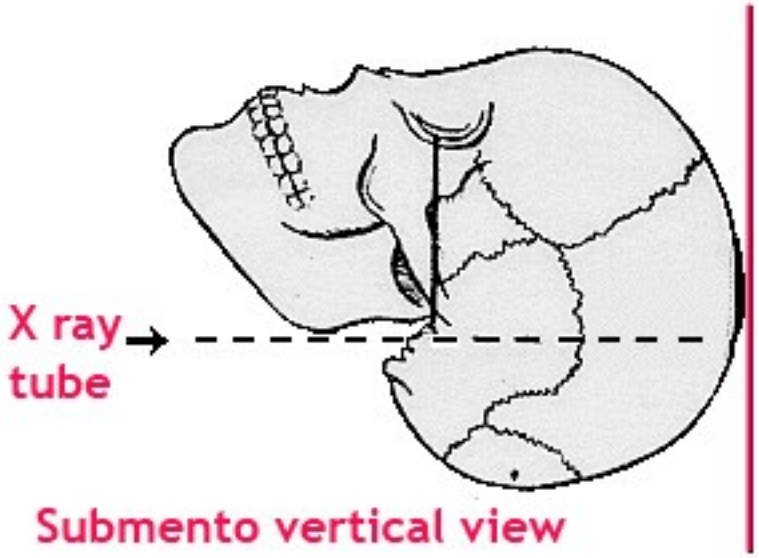
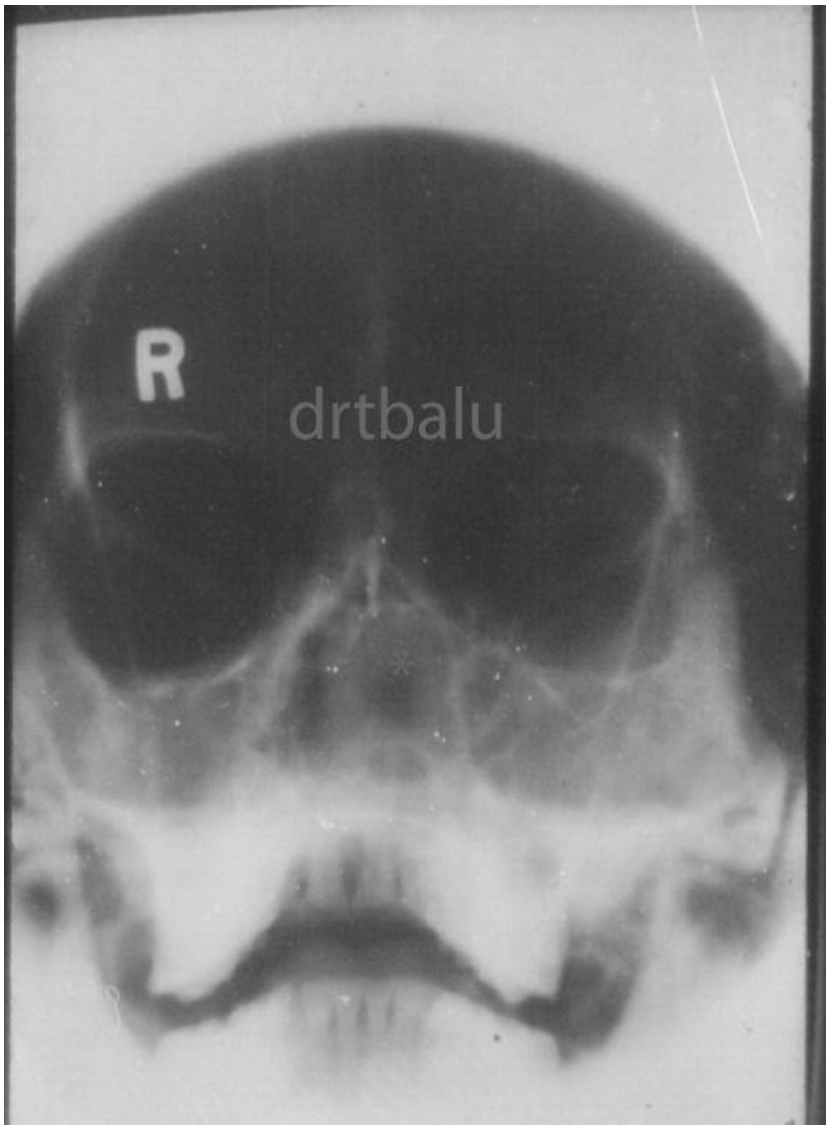


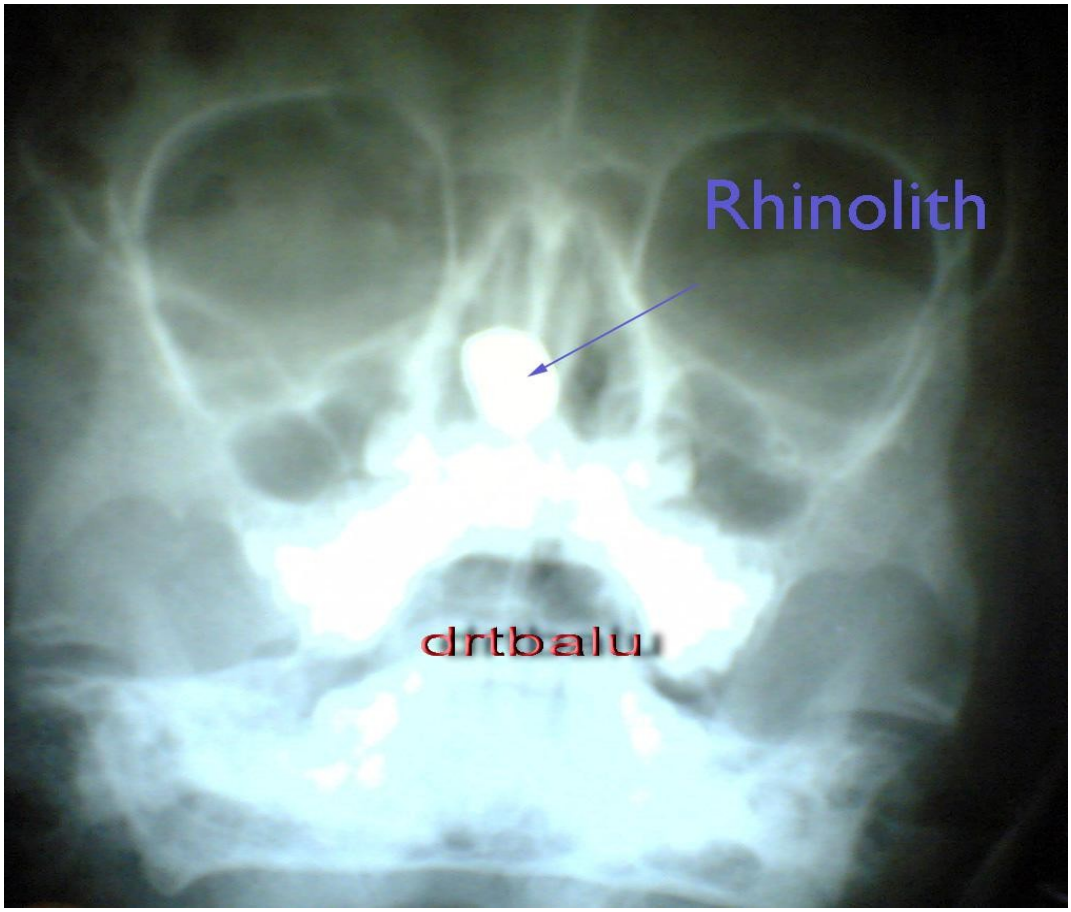
Figure showing positioning of patient for submento-vertical view

Fluid levels in sphenoid sinus can be clearly seen in this view. Zygomatic arch can also be clearly delineated in this view. This view is not popular these days because CT scans give better resolution of this area. CT scans have largely replaced this view.

Some pathological conditions that are clearly seen in sinus radiographs:



In the x-ray shown above, a swelling could be seen in the nasal septal area. Gas shadow could also be seen inside the swelling. This gas shadow could be caused by gas released by pyogenic organisms. The cartilaginous portion of the nasal septum is not visible, could have been eroded. Probable diagnosis is septal abscess. Both the maxillary sinuses also appear hazy due to infection. Collection of pus between the Perichondrium and the nasal septal cartilage compromises the nutritional status of the cartilage as it is dependent on the Perichondrium for its nourishment. This cartilage undergoes necrosis. Septal abscess in this patient could have been caused by chronic sinus infection (i.e. Note haziness of both maxillary sinuses in this patient).



X-ray PNS showing radio – opaque shadow inside the nasal cavity possibly rhinolith

X-ray lateral view:

This view helps in diagnosing pathologies involving frontal sinuses. It helps in determining whether the loss of translucency is due to thickening of the anterior bony wall or infection of the frontal sinus per se. This view also demonstrates fluid levels in the antrum. This view also gives information on the naso pharynx and soft palate. This is infact a standard projection used to ascertain enlargement of adenoid tissue.

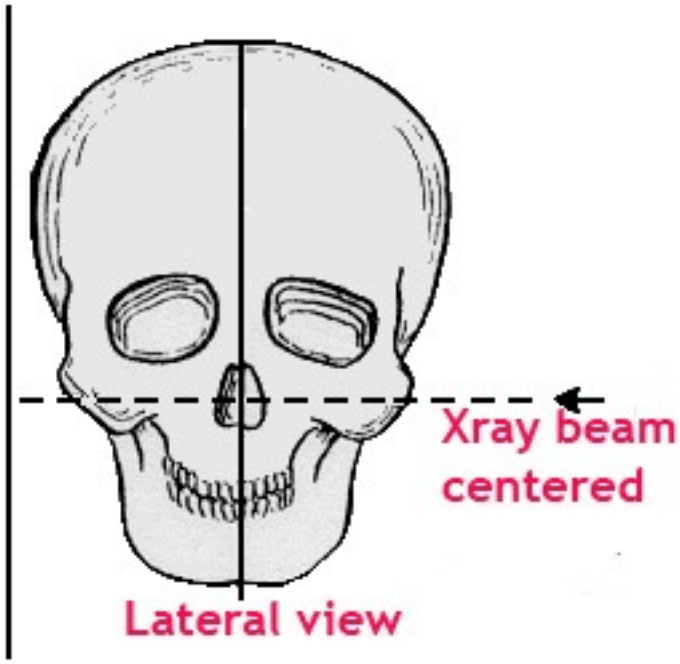
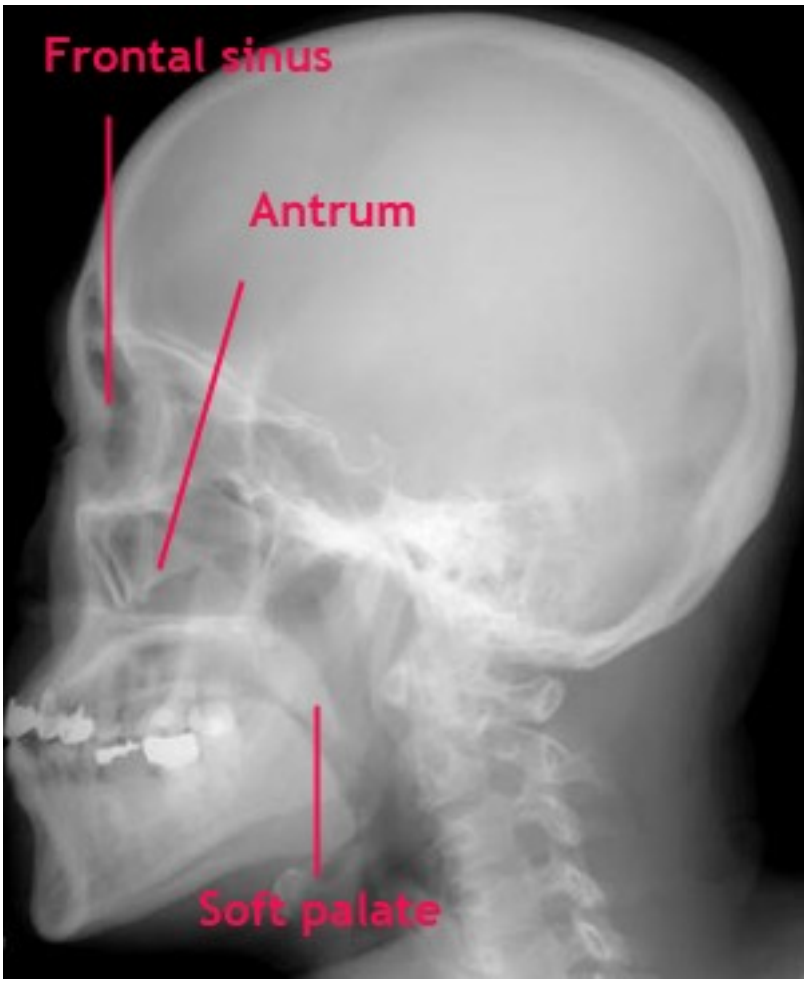


Figure showing skull position for xray skull lateral view

For this view the patient is made to sit with the sagittal plane parallel to the xray film and the radiographic base line is horizontal. The incident ray is horizontal and the incident beam is centered at the mid point of the antrum.



Xray skull lateral view

Oblique view:

This view helps in demonstrating posterior ethmoid air cells and optic foramen. To obtain this projection the patient is made to sit facing the film. The head is rotated so that the sagittal plane is tured to an angle of 39 degrees. The radiographic base line is at an angle of 30 degrees to the horizontal. The incident beam is horizontal and is centered so that the beam passes through the centre of the orbir nearest to the film.

X-ray nasal bone:

This is usually taken to rule out nasal bone fractures. One should be cautioned against misinterpreting groove for nasociliary nerve which could mimic fracture. Fracture nasal bones is actually a clinical diagnosis based on the following findings:

1. Tenderness over nasal bone area
2. Crepitus over nasal bone area
3. Swelling and deformity over nasal bone area

X-ray nasal bones showing fracture line

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